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OAT BRAN CONCENTRATE BREAD PRODUCTS: LONG TERM EFFECTS ON DIABETIC CONTROL

BY

© MARY E. PICK

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of **MASTER OF SCIENCE**

IN

FOODS AND NUTRITION

DEPARTMENT OF AGRICULTURAL, FOOD AND NUTRITIONAL SCIENCES

EDMONTON, ALBERTA

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UNIVERSITY OF ALBERTA

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The undersigned certify that they have read, and recommended to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **OAT BRAN CONCENTRATE BREAD PRODUCTS: LONG TERM EFFECTS ON DIABETIC CONTROL** submitted by **MARY E. PICK** in partial fulfillment of the requirements for the degree of **MASTER OF SCIENCE** in **FOODS AND NUTRITION.**

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ABSTRACT

Foods high in soluble fibre are recommended for the diabetic patient to improve glycemic response and lower plasma lipids. The long term effects of incorporating oat bran concentrate (OBC) bread products in the usual dietary pattern of non-insulin-dependent diabetic (NIDDM) subjects were evaluated via dietary, clinical and biochemical methods. Palatable high fibre OBC bread products were developed using OBC from Finland with a soluble fibre (ß-glucan) content of 22.8%. Eight NIDDM men (39 - 57 years), living in the community, participated in a 24-week study; a crossover experimental design with two periods (12 weeks each). Four randomly chosen subjects ate the OBC breads first; the remainder ate the control white bread (WB) first. Dietary intake was assessed via four 48-hour dietary recalls in each period. Blood glucose and insulin, via 8-hour profiles, and fasting lipid parameters were measured at 0, 12 and 24 weeks.

Total energy and macronutrient intakes were similar in both dietary periods. Mean total dietary fibre intake was 19 g per day in the WB period and 34 g per day (9 g soluble fibre per day from OBC) in the OBC period. Body weight remained stable. Mean dietary fat intake was 31% of total kcal in the WB period and 28% in the OBC period (NS). Mean glycemic response and insulin response areas (AUC) were lower ($p \leq 0.05$) for the OBC period than the WB period. After breakfast, OBC

period AUC was lower for glucose ($p \leq 0.01$) and insulin ($p \leq 0.05$); insulin peak was reached earlier ($p \leq 0.05$) than in the WB period. For insulin, total area under the curve was negatively correlated ($p \leq 0.05$) with both total and soluble dietary fibre intake. Mean total plasma cholesterol (C) and LDL-C were lower ($p < 0.01$) for the OBC period (4.56 and 2.59 mmol/L, respectively) than for the WB period (5.30 and 3.36 mmol/L, respectively). Mean HDL-C did not differ between the two periods; however, in the OBC period mean LDL-C/HDL-C ratio was reduced by 25% ($p \leq 0.05$). OBC bread products were well accepted.

Results indicate that incorporation of OBC bread products (9 g of soluble fibre) in the diet improved glycemic, insulinemic and lipidemic responses. Ingestion of OBC bread products could greatly benefit the overall health of non-insulin-dependent diabetic individuals.

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INTRODUCTION

Non-insulin-dependent diabetes is a major health problem in North America leading to serious complications such as cardiovascular disease, blindness, kidney failure and limb amputations. Control of blood glucose fluctuation by diet is essential to minimize development of diabetic complications. Recommendations for diabetic diets emphasize increased complex carbohydrate and dietary fibre. However, the amount and type of carbohydrate that should be incorporated into the diet remains controversial. A fibre intake of 40 g per day recommended by ADA (American Diabetes Association, 1992) is hard to achieve long term since the mean dietary fibre intake of North American males is only ~14 g per day.

Cereals, the main carbohydrate source in the North American diet provide insoluble fibre, not the soluble fibre advised for diabetic patients to improve glycemic response and normalize plasma lipids. Clinically, soluble fibre has been given as a supplement; however, incorporation of soluble fibre into acceptable foods (bread, buns, muffins) may improve long term dietary adherence. Oats and oat bran concentrate provide soluble fibre, specifically ß-glucan, that may enhance fibre intake long term. Oat bran food products have not been used in long term human studies. Assessments of the impact of oat

fibre on glucose/lipid parameters in diabetic subjects are lacking.

The objective of the present study was to assess the long term effects of incorporating oat bran concentrate bread/bread products in the usual dietary pattern of free-living non-insulin-dependent diabetic (NIDDM) men by dietary, clinical and biochemical methods.

LITERATURE REVIEW

DIABETES, CARBOHYDRATE INTAKE AND METABOLIC CONTROL

In North America one in 20 people (5% of the population) have diabetes (Joslin Diabetes Center, 1988). The majority of subjects have diabetes with onset as adults, non-insulin-dependent diabetes (NIDDM), ketosis resistant, Type II diabetes. Only a small minority, about 5 to 10%, have insulin-dependent diabetes (IDDM), ketosis-prone, Type I diabetes. Type II diabetes is characterized by an abnormality in glucose metabolism (Anderson, 1988). Achievement of physiological levels of blood glucose is the primary goal in diabetes management (Anderson et al., 1987; Canadian Diabetes Association, 1989; American Diabetes Association, 1992). To attain the best possible metabolic control, treatment of Type II diabetes relies heavily on diet therapy (Coulston, 1994). However, the ideal composition of a diabetic diet remains uncertain (American Diabetes Association, 1994) and the amount and type of carbohydrate that should be incorporated into the diet remains controversial.

The pathophysiology of non-insulin-dependent diabetes (NIDDM) involves both relative insulin deficiency and impairment of insulin action. The NIDDM subject has reduced glucose tolerance and is unable to remove a glucose load from his plasma within a specified time period. Gannon and Nuttall (1987) observed that, following consumption of a 50 g glucose

load, NIDDM subjects required four to five hours (compared to 1.5 hours for non-diabetic individuals) for postprandial glucose values to return to fasting levels, and that insulin concentrations were still slightly above fasting levels at five hours. The concentration of glucose in the blood is the result of the balance between net hepatic glucose uptake and peripheral glucose utilization. Each of these processes is regulated by a series of hormonal interactions in which insulin acts to lower blood glucose and several counter-regulatory hormones (glucagon, catecholamines, glucocorticoids, growth hormone) act to raise it.

The usual main stimulus of insulin secretion is a rise in the arterial blood sugar concentration. The ingestion of carbohydrate produces a prompt increase in plasma insulin and a decrease in glucagon concentrations. However, the change in blood glucose level after meals is not the only factor regulating plasma insulin increase. The rise in insulin occurs before the rise in arterial glucose concentrations. Early insulin release begins with the absorption of glucose; gastrointestinal factors ensure immediate release of insulin and accelerate glucose disposal, preventing excessive fluctuations of peripheral blood sugar levels. This entero-insular axis is an area of investigation; thus, the impact of gut hormones, such as cholecystokinin and gastric inhibitory peptide (GIP) is being evaluated (Morgan et al., 1979). This additional stimulus of gut hormones on insulin release has a

profound influence on the disposal of a glucose load and hence on glucose tolerance (Unger et al., 1967; Unger and Eisentraut, 1969).

Several dietary factors stimulate a rise in gut hormones: glucose, amino acids, proteins, fat and possibly non-glucose carbohydrates. Amino acids are potent stimulators of insulin release. The exact means by which amino acids stimulate the release of insulin is not clear; and probably more than one mechanism is involved since patients who respond to intravenous infusion of leucine may not respond to other amino acids which are known to stimulate insulin release (Floyd et al., 1966). In addition, there are differences in insulin response when NIDDM subjects are compared with non-diabetic young subjects, including: an abnormality in the usual biphasic response of insulin secretion; an increase in the ratio of proinsulin to insulin; a decrease in the response to amino acids; a failure of the glucose tolerance curve to return to normal by the second or third hour (Nuttall and Gannon, 1991).

Skeletal muscle is the predominant tissue for disposal of an oral glucose load. Insulin resistance is characteristic of NIDDM diabetes with insulin working ineffectively in the target tissues (muscle and liver). Hyperinsulinemia develops but there is hyperglycemia postprandially. One theory holds that with time the ß-cells become "exhausted" (Rossetti et al., 1990). Fasting and postprandial hyperinsulinemia are

indicators of insulin resistance. Several different mechanisms at the cellular level may impair sensitivity to insulin (Smith, 1994).

Guidelines for the nutritional management of diabetes in the 1990's state that 55 - 60% of total kcal be consumed as carbohydrate with foods high in fibre and unrefined carbohydrate eaten whenever possible. Dietary fibre intakes of up to 40 g per day or 25 g/1000 kcal are recommended (American Diabetes Association, 1992; Canadian Diabetes Association, 1989). The Canadian Diabetes Association (1989) has recommended increases in dietary carbohydrate to come from "unrefined starchy foods such as whole grain cereals and breads, legumes and tubers".

Dietary fibre refers to the non-starch polysaccharides and lignin present in plant products which are either very poorly digested or not digested by upper gastrointestinal (GI) tract enzymes (Southgate, 1977; Trowell, 1978; Theander et al., 1993). Estimates of daily total dietary fibre intakes in the US population range from 3 to 30 g per day (American Diabetes Association, 1992; Walker, 1993; Nuttall, 1993); the mean dietary fibre intake of US adults is estimated to be 13 g per day for ages 30 - 54 (Block and Lanza, 1987). Vegetables were the leading source of fibre, contributing 28% of total fibre. Fruits and legumes contributed 17% and 14%, respectively. The contribution of the cereal group was 19% for breads and 5% for breakfast cereals (Block and Lanza, 1987).

Estimates of fibre intakes of 19 g per day (half cereal-derived fibre) for Canadian males, and ~20 g per day for British individuals have been made (Kay, 1982). Therefore, for North American diabetic males, a daily fibre intake of 35 to 40 g per day could be difficult to realize on a long term basis.

Dietary fibre may be classified as water soluble or water insoluble. Soluble fibres are non-starch polysaccharides readily dispersed in water. Soluble, but not insoluble, fibres have demonstrated significant favorable effects on postprandial blood glucose and insulin values (Trowell, 1978; Anderson and Chen, 1986; Canadian Diabetes Association, 1989; American Diabetes Association, 1992). Anderson and Bridges (1988) analyzed soluble fibre composition of major food groups. Soluble fibre content, as percentage of total dietary fibre, averaged 32% for cereals, 32% for vegetables, 25% for dried beans and 38% for fruits. An additional dietary source of soluble fibres is polysaccharide food additives, e.g. pectin, gums, mucilages and algal polysaccharides. These products serve functional purposes in food systems by modifying the textural characteristics of foods. It has been estimated that the intake of soluble fibre as food additives is relatively low, about 2% of soluble fibre intake (Southgate, 1985).

Jenkins et al. (1976) showed that the addition of the soluble fibre, guar or pectin, to standardized meals resulted

in significant reduction of postprandial glucose in NIDDM, IDDM and in non-diabetic subjects. In normal and diabetic volunteers Jenkins et al. (1976, 1977) found that blood glucose levels were significantly lower following guar ingested in a glucose drink compared to glucose alone (Jenkins et al., 1976, 1977). Consumption of guar crispbread (14 - 26 g guar per day) by nine diabetic subjects reduced insulin requirements and reduced glycosuria 68% compared to a wheat bran control (Jenkins et al., 1978b). Other researchers confirmed Jenkins' (1976, 1977) results by demonstrating that soluble fibre consumption via guar supplementation resulted in lower postprandial glucose values in NIDDM subjects compared to no supplementation (Goulder et al., 1978; Aro et al., 1981; Ray et al., 1983; Najemnik et al., 1984). However, some criticisms of these studies included: no diet records were kept (Aro et al., 1981), diabetes was poorly controlled (Ray et al., 1983), carbohydrate content of the diet changed (Goulder et al., 1978; Ray et al., 1983) or subjects lost weight (Jenkins et al., 1988c). The level of dietary fibre in the diet which results in favorable metabolic response in NIDDM individuals is controversial. Dietary fibre supplementation may be more effective at higher levels of carbohydrate intake. In diabetic subjects, a mean reduction of 64% in glycosuria resulted when 14 - 24 g soluble fibre (guar) per day was taken with a carbohydrate intake over 40%; no reduction resulted with guar supplementation on lower

carbohydrate intakes (Jenkins et al., 1980d). In contrast, in six NIDDM subjects, Hollenbeck et al. (1986a) found no decrease in postprandial glucose values when fibre in the diet was increased from 11 to 27 g/1000 kcal per day. The fibre content of the diets in Hollenbeck's study was a realistic amount for everyday consumption. Thus carefully controlled studies with practical levels of fibre in the diet are required to determine the effect of fibre on postprandial glucose levels in NIDDM individuals.

Fibre supplementation in the form of guar is neither particularly palatable nor acceptable on a long term basis. Guar supplementation has not become widespread because, at levels required for effectiveness (10 to 15 g per day), it is unpalatable (Edwards et al., 1987). In addition, unpleasant side effects such as flatulence, bloating, diarrhea, increased number of stools have been reported by subjects ingesting guar (Aro et al, 1981; Ray et al, 1983; McIvor et al., 1985; Najemnik et al., 1984; Holman et al, 1987). Najemnik et al. (1984) reported that guar treatment had to be discontinued in 11% (10 patients) due to gastrointestinal side effects. Braaten et al. (1991) found that glucose and insulin responses to oat and guar gum meals were nearly identical; however, oat gum was more palatable. Anderson et al. (1991) suggest that oat fibre may have a beneficial effect on glucose tolerance in individuals with Type II diabetes. However, the researchers noted that the significant metabolic improvements obtained in

the short term hospital phase did not persist in the subsequent long term (10 week) home phase. This finding reaffirms the necessity of evaluating the incorporation of high mucilaginous fibre bread products in the usual dietary pattern of the individual with Type II diabetes for an extended period; the pilot study we propose will provide valuable data.

Oat is an excellent source of soluble fibre; the main component is a linear polysaccharide, the mixed-linked (1→3) (1→4) ß-D-glucan (Wood et al., 1989a, 1991; Autio et al., 1992a, 1992b; Theander et al., 1993). The fact that water-soluble mixed-link ß-glucans can produce highly viscous solutions is thought to be the reason for the beneficial effects of oat bran (Chen et al., 1981; Jennings et al., 1988; Wood et al., 1989a, 1990; Theander et al., 1993). Numerous studies have examined the effect of oat fibre as oatmeal or oat bran incorporated into the diet via supplements or various food products: hot or cold cereal, biscuits or muffins. Others have distributed oat bran sachets with study-specific recipes (Denmark-Wahnefried et al., 1990; Davidson et al., 1991; Leadbetter et al., 1991; Mackay and Ball, 1992; Uusitupa et al., 1992). These methods are not conducive to consumption of oat bran concentrate on a regular basis or for an extended period of time. Studies that have examined the effects of oat bran concentrate on metabolic parameters are lacking. Braaten et al. (1991) examined the effect of oat gum in a drink. Oat

bran and oatmeal contain 14.9 g and 9.9 g total dietary fibre, respectively, and 7.2 g and 5.0 g soluble fibre, respectively, per 100 g (Anderson and Bridges, 1988; Glore et al, 1994). Concentrating the soluble fibre from oats would reduce the amount required to be effective (Torronen et al., 1992).

Malkki et al. (1992b), of Exavena Oy/Inc., Espoo, Finland, developed a process (a combination of dry- and wet-milling operations) to remove non-fibre components, concentrate the soluble fibre in oat bran and lower the bulk to be consumed so that the amount to be eaten daily would be more realistic. The ß-glucan content is concentrated 2- to 3-fold from the starting level in the oat bran after the dry milling steps. The typical composition of oat bran concentrate made in an ethanol-water wet-milling process is 36 - 40% total dietary fibre and 15 - 20% ß-glucan. In Canada oat bran concentrate is available from the POS Pilot Plant in Saskatoon. Incorporation of concentrated oat bran in palatable food products for long term consumption could increase the soluble fibre content of the diet. Ranhotra et al. (1990) demonstrated that oat bran concentrate was more effective than oat bran if fed to rats at the same level. Only one published study (Torronen et al., 1992) has examined the effect of oat bran concentrate in food products in the diet. Oat bran concentrate was incorporated into a bread leavened with baking powder (Torronen et al., 1992). Development of acceptable food

products incorporating the concentrated form of oat bran for use in the daily diet is required.

Bread, which is eaten daily in North America, is a popular convenience food with potential for fibre enrichment. Estimates of bread consumption in North America are: 22.39 kg per capita in the United States (recent official consumption survey) and 65.18 kg per capita in Canada (Flax Council of Canada, 1994). Bread consumption in Canada appears to be much higher than in the United States; the estimate was based on weekly food expenditures in Canada. The average quantity of bread purchased per week per Canadian household was estimated to be 1.25 kg. The implication of dietary fibre in prevention of certain diseases has helped increase demand for specialty breads (Krishnan et al., 1987).

Few published studies have examined the metabolic effects of oat fibre incorporated into acceptable yeast breads and no published studies are available which examine oat bran concentrate incorporated into yeast breads and baked products in the diet. DeGroot et al. (1963) found that, in 21 healthy male volunteers who substituted bread containing 140 g of oatmeal per day for the usual bread in the diet for 3 weeks, total blood cholesterol was 11% lower. However, no details about the oatmeal bread were published. Two researchers (Kestin et al., 1990; Bremer et al., 1991) examined metabolic effects of oat bran in yeast breads. Few details of the bread were published. In the Bremer et al. (1991) study the oat

bread, supplied by a commercial bakery, contained 1.2% soluble fibre and contributed 40% of the total daily fibre intake for the subjects. Torronen et al. (1992) examined the effects of baking powder leavened bread containing 11.2 g ß-glucan from oat bran concentrate compared to the usual bread in the diet.

Bread is a product in which quality characteristics are markedly affected by ingredients. Since the addition of fibre is detrimental to bread quality, incorporation of large amounts of fibre into yeast breads presents challenges. Many studies illustrate the importance of determining the appropriate levels at which fibre ingredients can be successfully incorporated. Problems include poor texture and off-flavor. Klopfenstein and Hoseney (1987) developed yeast breads with 7% and 13% oats ß-glucan. The texture of the 7% ß-glucan bread was similar to multi-grain breads presently on the market, while the higher fibre 13% ß-glucan bread was very dense with a dark brown crust (Klopfenstein and Hoseney, 1987). The addition of more than 7 - 10% of any fibrous material results in major changes in quality characteristics of bread, requiring changes in processing techniques and reformation or reworking of doughs (Navickis and Nelsen, 1992). Fibre incorporation into bread dough reduces some desirable textural properties (unacceptable loaf volume, crumb texture and color and mouthfeel) of the final product. Dough quality deteriorates above 10% fibre supplementation and is unacceptable above 20% (Navickis and Nelsen, 1992). As more

fibre is added, dough strength and extensibility decrease (Navickis and Nelsen, 1992). Krishnan et al. (1987) found that, with oat bran substitution, as the level of oat bran increased, loaf volume decreased. Oat bran bread quality was influenced by gluten dilution and gums present in the oat bran. In addition, the amount of water required in oat bran dough increased as oat bran level increased (Krishnan et al., 1987). Shogren et al. (1981) found that the deleterious effects of 15 percent wheat bran added to wheat flour could be counteracted by adding vital wheat gluten plus a surfactant alone or in combination with shortening. Adding 0.5 g surfactant in combination with 3 g shortening per 100 g mixture produced improvements in bread volume and crumb grain above the levels produced when shortening or surfactant were used individually (Shogren et al., 1981). For oat bran bread, mixing time of dough increased over the control, stickiness was encountered during the mixing process, and inclusion of bromate in the formula improved loaf volume, grain and texture of the oat bran bread (Krishnan et al., 1987).

Little is known about the physiological properties of many complex carbohydrates in foods. Jenkins et al. (1981) developed the glycemic index to measure the ability of a carbohydrate to contribute to the concentration of blood glucose. A high glycemic index indicates that the dietary carbohydrate elevates blood glucose faster than a carbohydrate of a lower glycemic index. Jenkins et al. (1981) reported that

the lowest glucose responses were obtained with legumes, which are high in dietary fibre. The importance of viscosity in regulating postprandial glucose and insulin levels was first suggested by Jenkins et al. (1978a). A flattening of the peak rise in glucose concentration was found to be greater with guar (soluble fibre) than wheat bran (insoluble fibre); reduction in mean peak rise in blood glucose concentration for each substance tested correlated with its viscosity. When hydrolyzed non-viscous guar was used, flattening of the glucose response did not occur (Jenkins et al., 1978a). Viscous substances may act through delayed gastric emptying and delayed absorption of glucose from the small intestinal lumen. Tinker and Schneeman (1989) demonstrated that highly viscous substances in the intestine slow digestion through decreased access of digestive enzymes. Reduced glucose diffusion may result from increased unstirred layer thickness or an impairment in convective movement in the intestinal lumen (Nuttall, 1993). However, Braaten et al. (1991) found that both guar gum and oat gum similarly reduced the postprandial glucose rise although viscosities of the two gums differed. Edwards et al. (1987) also found that glycemic response between gums of varying viscosities was not significantly different. In this case differences in viscosity did not affect blood glucose response. Further study of the effects of highly mucilaginous fibres such as oat bran

concentrate in slowing glucose absorption in NIDDM subjects is needed.

Other possible mechanisms for the effects of fibre on glucose absorption are appearing in the literature. Tappy et al. (1986), for example, suggest that the glycemic response obtained from legumes may be related to the histological structure of the thick-walled cells which prevent the complete swelling of starch during cooking and decreases access of digestive enzymes. Intact plant cell walls are rich in unavailable polysaccharides which can serve as a barrier to the penetration of digestive enzymes. It is difficult to identify components in intact cell walls. The diversity of constituents includes dietary fibre components that exist in a matrix and are linked with other carbohydrates, proteins, fats and inorganic compounds.

Hollenbeck et al. (1986b) suggested that the glycemic index was inadequate to evaluate the physiological impact of dietary components on blood glucose response. The criteria they established to evaluate the metabolic impact of carbohydrate containing foods was that the metabolic response be estimated in both long-term and short-term studies and that the response be defined in individuals for whom dietary recommendations are proposed. Coulston et al. (1987) compared plasma glucose and insulin responses to mixed meals of high-, intermediate- and low-glycemic potential. They concluded that the rise in blood glucose is not precisely predictable and is

subject to many variables including the nature of the food eaten. Crapo et al. (1976) showed that the nature of the starch in food influences glycemic response. The rise in blood glucose after ingestion of bread, corn or rice was lower than after ingestion of potato or glucose. It is known that a proportion of dietary starch can escape digestion in the human gastrointestinal tract. This portion reaches the colon where it can be fermented by bacteria in a way that is similar to dietary fibre. This resistant starch has been estimated to comprise 7 - 10 % of the carbohydrate from oats and wheat, 20% from baked beans, and less than 1% from rice (Levitt et al., 1987). There are various reasons why starch escapes digestion including:

- gelatinization of starch formed with moist heat (e.g. bread making) results, on cooling, in molecules (retrograded amylose) which are resistant to hydrolysis by the α-amylase enzymes in the intestine.
- physical inaccessibility to digestive enzymes (α-amylase), e.g. partly milled grains, seeds.
- α-1→6 linkages of amylopectin. α-amylase in the human intestine hydrolyzes 1→4 glycosidic bonds not 1→6 bonds.

In addition, plant foods contain non-carbohydrate constituents which may contribute to the properties influencing glycemic response. Such non-carbohydrate compounds include: Maillard reaction products, digestive enzyme inhibitors, cell wall glycoproteins and phenolic esters.

Hollenbeck et al. (1986b) suggested that an evaluation of the glycemic response to foods was not complete without an evaluation of the insulin response. Bornet et al. (1987) examined both glycemic and insulinemic indices of six starch-rich foods taken alone and in a mixed meal by NIDDM subjects. These researchers found that the glycemic responses to bread ingestion were variable and greater than the indices obtained by Crapo et al. (1981) or Jenkins et al. (1981). The insulinemic indices were also greater than those obtained by other researchers. They suggested possible reasons for the differences including differences in bread processing and differences in the origin of the wheat. Bornet et al. (1987) indicated that for the calculation of the glycemic index the reference food should not be white bread as Jenkins uses, but rather glucose which gives consistent values.

Controversy surrounds the usefulness of the glycemic index (Wolever et al., 1991) to evaluate physical properties of dietary carbohydrate. Several researchers contend that the glycemic index of a mixed meal may be predicted from the glycemic index of the component carbohydrate foods (Wolever et al., 1985; Chew et al., 1988; Indar-Brown et al., 1992). Others, however, find that the glycemic index may aid in predicting the magnitude of glucose excursions when single foods are eaten alone, but the glucose response to whole meals is more complex (Nathan, 1987). Many components of whole meals, including dietary fibre, affect glycemic response. The

non-carbohydrate portion of the diet affects glycemic response. Highly significant correlations have been found between glycemic index (and area under the curve) and dietary protein and fat (Hollenbeck et al., 1986b). Researchers (Collier et al., 1984) found a smaller glycemic response with fat added to a carbohydrate meal than without. In seven healthy subjects, postprandial glucose levels were lowered significantly following the addition of 37.5 g fat to a meal containing 75 g carbohydrate (Collier et al., 1984). Fat may affect glycemic index by delaying gastric emptying (Collier and O'Dea, 1983; Collier et al., 1984) or by reducing starch gelatinization (Hoseney et al., 1977). Collier et al. (1984) determined both glycemic and insulinemic indices. Fat, co-ingested with carbohydrate, potentiated the insulin response. Others have demonstrated that insulin sensitivity is inversely related to the fat content of the diet (Himsworth, 1935; Beck-Nielsen et al., 1978). When protein is ingested with glucose there is a potentiation of the insulin response. For some NIDDM individuals the glycemic response is lower for protein and glucose than for glucose alone (Gannon et al., 1988). There is no simple correlation between the fibre content and the glycemic index of food (Jenkins et al., 1981). Different types of dietary fibre have varying effects.

Numerous questions have been raised about methodology used to study glycemic effects as illustrated by the work of two groups of researchers, Jenkin's group and Hollenbeck's

group, disagree on the efficacy of high carbohydrate and high soluble fibre dietary levels on glycemic response. The following differences in the respective studies must be considered:

1. Method of fibre administration: Many of Jenkin's studies have examined fibre supplementation either compared to a glucose drink or in a drink accompanying the meal. The Hollenbeck group examined whole meals containing fibre rich foods. Whole meals with easily obtainable fibre rich foods are most likely to be consumed by individuals on a long term basis.

2. Time of day: It is known that time of day of the test affects glycemic response; diurnal variations occur in oral glucose tolerance (Jarret et al., 1972). In 24 non-diabetic subjects following glucose tolerance tests, mean blood glucose was significantly lower in the morning than in the afternoon or evening; however, plasma insulin levels were highest in the morning test (Jarret et al., 1972). Test meals in the Coulston et al. (1984) study were taken at 12 noon while test meals in the studies of Jenkins et al. (1976, 1980a, 1981, 1982) were taken at 800 hours.

3. Methodological differences: For calculations of area under the curve, Jenkins' group (Jenkins et al., 1983a; Wolever et al., 1985; Wolever and Jenkins, 1986, Jenkins et al., 1988a) utilize the incremental area under the curve, whereas

Hollenbeck's group (Hollenbeck et al., 1986a; Coulston et al., 1981, 1987) use the total area under the curve.

Researchers have used several methods, total, incremental and positive area under the curve, to study glycemic response. LeFloch et al. (1990) reported that total area under the curve was strongly correlated to basal blood glucose whereas incremental and positive area under the curve were strongly correlated to each other and better described glycemic response to foods. For estimating incremental area under the curve controversy remains whether area below fasting (baseline) level should be subtracted or, ignored and replaced by zero (LeFloch et al., 1990). Positive area under the curve is computed with information derived from sample points to find the point where the curve cuts baseline; positive area under the curve may be a more reliable estimation of area above baseline (LeFloch et al., 1990).

Insulin responses are important when evaluating physiological responses to food. In studies using the soluble fibre guar, Jenkins et al. (1982) suggested that postprandial insulin response can be predicted by post meal glucose response and that insulin and glucose are similarly lowered following ingestion of soluble fibre. In NIDDM subjects, both postprandial glucose values and insulin response were 50% lower following soluble fibre ingestion (Jenkins et al., 1978a, 1980a). However, it has been demonstrated that some meals that give similar glucose responses do not give similar

insulin responses and that non-carbohydrate dietary components affected response (Chew et al., 1988). Several researchers have demonstrated improved insulin response following ingestion of high carbohydrate, high fibre diets (Aro et al., 1981; Simpson et al., 1981; Kay et al., 1981; Ray et al., 1983; Karlstrom et al., 1984; DelToma et al., 1988a, 1988b; O'Dea et al., 1989).

In the literature there are reports of both successes and failures resulting from the use of high fibre diets in the treatment of diabetes. Anderson and coworkers (Kiehm et al., 1976) showed that the high carbohydrate, high fibre diet (HCF) was beneficial for the diabetic individual, resulting in lower glucose levels and lower insulin or hypoglycemic drug requirements. The HCF diet contained fibre-rich foods from a variety of food groups and provided over 60 g dietary fibre per day. Fibre sources included cereals (40% of total fibre intake), vegetables (51%) and fruits (9%). The HCF diet also lowered serum lipid levels. Anderson's group (Kiehm et al., 1976) reported that diets high in carbohydrate (70%) and fibre (14.2 g crude fibre) resulted in greater improvement in glycemic control than a traditional diet containing 43% carbohydrate and 4.7 g crude fibre. For the five NIDDM subjects, sulfonylurea drugs were discontinued. Anderson and Ward (1978) then used high carbohydrate, high fibre diets containing 70% carbohydrate and 35 g dietary fibre/1000 kcal. Compared to traditional diets, this high fibre diet improved

both fasting and postprandial glucose response and reduced the use of hypoglycemics by some subjects (Anderson and Ward, 1978). The HCF long term maintenance diets (60% carbohydrate, 20% fat and 25 g dietary fibre/1000 kcal) sustained improvement in glycemic control and insulin requirements for up to eight years. However, on HCF diets, patients lost weight (Kiehm et al., 1976; Anderson et al., 1984b; Nuttall, 1993) and weight loss is known to favorably influence blood glucose levels. Of the subjects monitored for four years, 75% adhered to the HCF maintenance diets well (Story et al., 1985; Anderson and Bryant, 1986). One of the excellent sources of dietary fibre in HCF diets was dried beans; beans in large quantities are not generally well accepted by North Americans. Another component of Anderson's diets was oats (Anderson and Chen, 1979; Anderson et al., 1984a, 1984b); oat is a good source of soluble fibre and is acceptable to the North American palate. Other researchers have demonstrated improvements in glycemic response in NIDDM subjects with consumption of high fibre natural foodstuffs (Kay et al., 1981; Simpson et al., 1981; Rivellese et al., 1983; Riccardi et al., 1984; DelToma et al., 1988a, 1988b; O'Dea et al., 1989). Several researchers have used extremely high levels (>50 g/1000 kcal) of dietary fibre (Simpson et al., 1981; Riccardi et al., 1984) to obtain improvements in glucose metabolism. However, the amount of fibre (~50 - 100 g dietary fibre per day) consumed by subjects in these studies may be

unrealistic on a long term basis for North Americans. In contrast, several well controlled studies where high carbohydrate high fibre diets containing fibre-rich foods were consumed reported no improvement of glycemic parameters in NIDDM subjects. Hollenbeck et al. (1986a) found no significant differences in day long plasma glucose and insulin responses following an increase in the dietary fibre content of the diet from 11 to 27 g/1000 kcal per day.

The successful dietary regimes for NIDDM individuals contain more soluble dietary fibre components than the unsuccessful dietary regimes. In 10 NIDDM subjects, DelToma et al. (1988b) reported that postprandial blood glucose response to low fibre (6.7 g total dietary fibre, 4.7 g insoluble, 2.0 g soluble), and high insoluble fibre (33.6 g total dietary fibre, 27.2 g insoluble, 6.4 g soluble) test meals was similar; however, a high soluble fibre (32.9 g total dietary fibre, 18.1 g insoluble, 14.8 g soluble) meal resulted in significantly lower glucose responses than the low fibre or high insoluble fibre meals. Several researchers have reported that, for NIDDM individuals, fibre enriched diets have produced significant reductions in fasting glucose values (Kiehm et al., 1976; Aro et al., 1981; Ray et al., 1983; Jenkins et al., 1988c). In contrast, other researchers found that, for NIDDM subjects, fasting blood glucose after dietary fibre supplementation was unchanged (Kay et al., 1981; Riccardi et al., 1984) or even increased (Jenkins et al.,

1980b; Hollenbeck et al., 1986a; Coulston et al., 1987). In NIDDM individuals fasting glucose concentration is very sensitive to reduction in total energy intake (Savage et al., 1979; Nuttall, 1993). In addition, weight loss favorably affects glycemic response (Wolever et al., 1992; Wing et al., 1994). Studies can be difficult to compare due to differences in experimental design and in type and amount of fibre added (Nuttall, 1993). Nuttall (1993) criticized some studies because the diet was not controlled. In some studies where glycemic improvements were seen, results were complicated by weight loss, a decrease in total food energy, changes in the total carbohydrate content of the diet, changes in the types of carbohydrate in the diet and the inclusion of large amounts of legumes (Nuttall, 1993). A few studies report deterioration of metabolic control of NIDDM subjects on high fibre diets. Both Coulston et al. (1987) and Scott et al. (1988) demonstrated deterioration in metabolic control in NIDDM subjects on high fibre diets (38 g and 35 g per day, respectively). In fact, in the Scott et al. (1988) study, subjects who ate the most fibre showed the greatest deterioration in metabolic control. Further well controlled long term studies of the efficacy of fibre in the diet on metabolic control of NIDDM subjects are needed.

Riccardi et al. (1984) examined further the effects of varying the amount of fibre in the diet as well as the amount of carbohydrate in the diet. Riccardi et al. (1984) showed

that increasing the amount of carbohydrate from 240 g to 360 g per day in the diet of 14 diabetic subjects had very little influence on fasting blood glucose concentrations and contributed to the deterioration of blood glucose control in the postprandial phase. However, parallel increases of dietary carbohydrate (42% → 53%) and fibre (16 → 54 g) produced significant improvement in postprandial glucose and daily blood glucose profile. The distribution of calories between carbohydrate and fat in the diet of the NIDDM individual is a controversial issue of current concern. For the NIDDM subject, carbohydrate tolerance worsens as insulin reserve declines; a high-carbohydrate diet may elevate glucose levels and may also contribute to abnormalities of lipoprotein metabolism. The new May 1994 nutrition recommendations for diabetic individuals (American Diabetes Association, 1994) state that nutrition treatment should be based on nutritional assessment and outcome goals and focus on the total amount of carbohydrate rather than the type. Further controlled long term studies are needed to evaluate both quality and quantity of carbohydrate in the diet of NIDDM subjects.

LIPIDS, DIABETES AND DIETARY FIBRE

For diabetic individuals, the aim of nutritional therapy is to achieve both near-normal plasma glucose levels and optimal plasma lipid levels (Anderson et al., 1987; Canadian Diabetes Association, 1989; American Diabetes Association,

1992). The primary cause of death in Type II (non-insulin-dependent) diabetes is atherosclerosis (Canadian Diabetes Association, 1981). Risk factors for atherosclerosis and cardiovascular disease are over-represented in diabetes (American Diabetes Association, 1992); diabetic individuals have at least four risk factors: hyperglycemia, hyperinsulinemia, hypertriglyceridemia and decreased HDL-cholesterol concentrations (Coulston, 1994). The lipid abnormalities associated with non-insulin-dependent diabetes are elevated blood triglycerides and decreased HDL-C; total-C and LDL-C levels are frequently in the normal range (DeFronzo and Ferrannini, 1991; Hollenbeck and Coulston, 1991).

Soluble fibres such as oat bran, beans, guar gum and fruit pectin have been reported to lower serum cholesterol (C) levels. Furthermore, soluble fibre selectively lowers low density lipoprotein cholesterol (LDL-C) levels, sparing or actually raising the cardioprotective high density lipoprotein cholesterol (HDL-C) fraction (Kirby et al., 1981; Anderson et al., 1984a, 1984b). Wheat bran and other sources of insoluble fibre have not been shown to lower serum cholesterol levels (Gold and Davidson, 1988). Researchers have studied the effects of soluble fibre on blood lipids in diabetic subjects using guar supplementation and natural foods (Rivellese et al., 1980; Jenkins et al., 1980a, 1980b; Aro et al., 1981; Najemnik et al., 1984; O'Dea et al., 1989). In NIDDM subjects, high carbohydrate high fibre diets resulted in lower total and

LDL-C and unchanged HDL-C and TG levels than high carbohydrate low fibre diets (Rivellese et al., 1983; Riccardi et al, 1984; O'Dea et al., 1989). A prolonged period of time (24 weeks) is required to increase HDL-C levels (Jenkins et al. 1988c).

Hypertriglyceridemia has been linked to increased risk of cardiovascular disease in diabetic individuals (Hollenbeck et al., 1986b; Hollenbeck and Coulston, 1991). A major concern is that high carbohydrate diets may cause hypertriglyceridemia (Anderson and Ward, 1978). Coulston (1994) concluded that high carbohydrate, low fat diets do not alter total-C or LDL-C levels but raise plasma triglyceride levels in NIDDM subjects. When carbohydrate was isocalorically substituted for fat in the diet, plasma triglyceride levels increased and HDL-C levels decreased (Garg et al., 1988; Coulston et al., 1987). However, Jenkins (1988) found that high carbohydrate, low fibre diets increased fasting serum triglyceride values but that high carbohydrate, high fibre diets did not raise serum triglyceride levels. Others have reported that high carbohydrate diets, which are high in fibre, were effective in lowering fasting triglyceride values in persons with hypertriglyceridemia (Anderson and Chen, 1979; Simpson et al., 1981; Anderson et al., 1987).

Research studies support the use of a soluble fibre, such as oat bran, as a cost effective alternative to drug therapy. In an estimate of "cost per year of life saved" as the result of treatment of high cholesterol levels, cost for oat bran was

$17,800 compared to $117,400 for cholestyramine or $70,000 for colestipol (Kinosian and Eisenberg, 1988). DeGroot et al. (1963) were the first researchers to report that blood cholesterol levels in humans were lowered by the addition of an oat product to the diet (Ripsin et al., 1992). Total blood cholesterol was 11% lower for 21 healthy males who substituted bread containing 140 g of oatmeal per day for the usual bread in their diet for three weeks (DeGroot et al., 1963). Since then, numerous researchers have studied the effect of oat products on lipid lowering in non-diabetic human subjects. Studies have demonstrated that oat products lower total-C and LDL-C in human subjects: Judd and Trusswell, 1981; Kirby et al., 1981; Anderson et al., 1984a, 1984b, 1987, 1990a, 1991; Storch et al., 1984; VanHorn et al., 1986, 1988, 1991; Turnbull and Leeds, 1987; Gold and Davidson, 1988; Kestin et al., 1990; Denmark-Wahnefried et al., 1990; Welch et al., 1990; Leadbetter et al., 1991; Davidson et al., 1991; Keenan et al., 1991; Uusitupa et al., 1992; Kashtan et al., 1992; Whyte et al., 1992; Lepre and Crane, 1992. In these studies HDL-C of subjects remained unchanged; blood triglycerides were unchanged or slightly lowered. No published studies have examined the effect of oat products in diabetic individuals. Several studies did not demonstrate hypolipidemic effects following consumption of oat products in the diet. These studies have used smaller amounts of ß-glucan (Bremer et al., 1991; Mackay and Ball, 1992), examined non-fasting blood

cholesterol (Saudia et al., 1992), or had initial lipid values in the normal range (Swain et al., 1990). Lack of an adequate control group (Anderson, et al., 1984a; Denmark-Wahnefried et al., 1990) is an additional consideration. Significant weight loss for subjects (Jenkins et al., 1980b; Anderson et al., 1984a, 1984b; Denmark-Wahnefried et al., 1990; Swain et al., 1990) may result in reduction of blood lipids and overshadow any reduction due to oat product consumption (Ripsin et al., 1992).

Studies examining the effect of oat products on blood lipids conducted in metabolic ward settings tend to show greater blood lipid reductions than experiments conducted in free-living individuals. Metabolic ward studies resulted in blood cholesterol reductions of 13% (Kirby et al., 1981), 19% and 24% (Anderson et al., 1984b; Anderson and Bryant, 1986); while research involving free-living individuals has produced more variable results, from no significant change (Leadbetter et al., 1991) to significant changes ranging from 2.2% (Keenan et al., 1991) to 12% (Storch et al., 1984).

Initial cholesterol level of subjects may be a factor influencing the amount of lipid lowering that may be achieved with soluble fibre consumption. In a meta-analysis Ripsin et al. (1992) found that trials examining subjects with initial cholesterol levels of ≥5.8 mmol/L and using ≥3 g soluble fibre per day displayed five-fold greater reductions in total-C than studies using subjects with lower baseline lipids and lower

levels of soluble fibre. VanHorne et al. (1991) noted a greater reduction in total-C in free living individuals with a baseline median serum total-C ≥6.34 mmol/L than in subjects with a baseline total-C ≤6.34 mmol/L. Anderson et al. (1991) fed 13.4 g soluble fibre per day to 20 subjects with an initial cholesterol level of 6.9 mmol/L in a metabolic ward situation and achieved a mean cholesterol reduction of 12.8%. Swain et al. (1990) demonstrated no lipid lowering in subjects with initial cholesterol levels of 4.8 mmol/L. In normocholesterolemic subjects reductions in serum cholesterol resulted when large amounts of fibre were included in the diet (Kiehm et al., 1976; Anderson and Chen, 1979; Anderson and Ward, 1978: Rivellese et al., 1980; Simpson et al., 1981). With oat bran consumption blood lipid lowering has occurred in both hyper- and normo-cholesterolemic subjects (Gold and Davidson, 1988). Further study of the efficacy of oat bran concentrate on blood lipids of Type II diabetic subjects is required.

The amount of fibre that should be incorporated into the diet to produce favorable metabolic changes remains controversial. In studies where over 3 g ß-glucan per day has been consumed, significant lipid lowering has occurred (Ripsin et al., 1992; Malkki, 1993). Ripsin et al. (1992), in a meta-analysis of 20 trials found a significant cholesterol lowering effect in trials where a large amount of ß-glucan (50 to 100 g oat bran per day) was ingested. An increase in soluble

fibre intake of ~3 - 6 g per day in a metabolic ward setting decreased serum total-C of hypercholesterolemic men by 5.4 - 12.8% (Anderson et al., 1990a, 1990b). Davidson et al. (1991) reported that ß-glucan had an independent hypocholesterolemic effect. The 156 subjects in these studies had LDL-C levels ranging from 3.37 to 4.14 mmol/L and also above 4.14 mmol/L. Fifty-six g oat bran resulted in a significantly greater reduction in LDL-C than 56 g oatmeal; there was a dose-dependent reduction in LDL-C levels. VanHorn et al. (1986) also showed that oats had an independent hypocholesterolemic effect. In normolipidemic, free living individuals 60 g per day rolled oats resulted in a 3% reduction in serum cholesterol in addition to a 5% reduction achieved by a low fat diet (VanHorn et al., 1986).

Interpretation of data among studies is made difficult because the amounts of soluble fibre used differs and different oat products have been utilized. Rolled oats (DeGroot et al., 1963; Judd and Trusswell, 1981; VanHorn et al., 1986) and oat bran (Kirby et al., 1981; Storch et al., 1984; Turnbull and Leeds, 1987; Gold and Davidson, 1988; Swain et al., 1990) have been used. The ß-glucan itself may differ because different strains of oats have been used or the nature and processing of the oat bran differs (Whyte et al., 1992). All studies do not state the amount of ß-glucan in the oat product evaluated (Anderson and Gustafson, 1988). Whole oats contain 25% total dietary fibre and 3% soluble fibre (Anderson

and Chen, 1979), oatmeal contains ~13.9% total dietary fibre and 7.7% soluble fibre while oat bran contains 27.8% total dietary fibre and 14.0% soluble fibre (VanHorn et al., 1986). The minimum level of ß-glucan in oat bran is 5.5% according to the American Association of Cereal Chemists (AACC) definition (AACC Committee, 1989). In addition, different methods have been used to determine the fibre content of foods; estimations of fibre content vary according to method of determination used (Schneeman and Tietyen, 1994). The Prosky procedure (enzymatic) is an official method of the Association of Official Analytical Chemists (AOAC) (Schneeman and Tietyen, 1994).

Mechanisms responsible for the hypocholesterolemic effect of oat bran and other soluble fibres are yet to be determined. It is thought that the main effective component of oats is ß-glucan; the role of tocotrienols or other components is thought to be small (Malkki, 1993). The lysine/arginine ratio (L/A) of the protein may influence lipid metabolism (Kritchevsky et al., 1987). Potential mechanisms of action of soluble fibre on lipid metabolism include: increased fecal excretion of bile acids and altered bile acid metabolism, slowed rate of lipid absorption, enhancement of short chain fatty acid production by polysaccharide fermentation in the large bowel and altered lipoprotein metabolism by increasing LDL receptors (Anderson et al., 1991; Schneeman and Tietyen, 1994). Cholesterol synthesis in the liver may be reduced by

lowering postprandial glycemia and insulinemia (Anderson et al., 1991; Brand Miller, 1994). Insulin regulates both cholesterol and triglyceride synthesis; fasting triglyceride reflects glucose control (Brand Miller, 1994).

DIABETES AND LONG-TERM METABOLIC CONTROL

Achievement of good metabolic control by means of diet is crucial in reducing long-term complications of diabetes as the Diabetes Control and Complications Trial has emphasized (American Diabetes Association, 1993). Control of blood glucose fluctuations by diet is essential to minimize development of diabetic complications (Anderson et al., 1987; Canadian Diabetes Association, 1989). A relationship exists between hyperglycemia and the development of small vessel disease (retinopathy, nephropathy, neuropathy). Recent work shows that chronic hyperglycemia also has a deleterious effect on ß-cell function; glucose toxicity has an impact on insulin secretion, insulin action and glucose transport (Rossetti et al., 1990). Glucose toxicity results in down regulation of the glucose transport system and a generalized desensitization of all cells in the body, playing a major role in the development of insulin resistance and impaired insulin sensitivity of the NIDDM subject (Rossetti et al., 1990). Insulin sensitivity improves as glycemic control is enhanced in the NIDDM diabetic individual (Yki-Jarvinen, 1992). Thus tight glycemic control is imperative for treatment of the NIDDM subject.

Brand et al. (1991) found that in 16 well controlled NIDDM subjects mean glycosylated hemoglobin was 11% lower ($p \leq 0.05$) following 12 weeks on a low glycemic index diet than after 12 weeks on a high glycemic index diet. However, long term human studies in which refined concentrated soluble fibre was added to the diet as guar (Holman et al., 1987; Beattie et al., 1988; Uusitupa et al., 1989) and pectin (Gardner et al., 1984) demonstrated that long-term glucose control, measured by glycosylated hemoglobin (King, 1987) was not improved with long term ingestion of 15 to 20 g dietary fibre per day. Therefore, no definite conclusions have been reached on the effect of dietary carbohydrates on metabolic control in non-insulin-dependent diabetes; the results reported in the literature are contradictory.

Oat bran concentrate food products have not been used in long-term human studies and assessments of the impact of oat bran concentrate on glucose and lipid parameters in diabetic subjects are lacking. Furthermore, the food industry has been encouraged to intensify efforts to develop and market foods that will make it easier for individuals to achieve a higher fibre intake (Klopfenstein and Hoseney, 1987). The present study was undertaken to examine the long-term effects of oat bran concentrate incorporated into acceptable bread/bread products as a dietary staple on the glycemic, insulinemic and lipid parameters of free living NIDDM diabetic men.

METHODOLOGY

The study was conducted from August, 1992 to May, 1993. The protocol was approved by the Ethics Review Committee of the Faculty of Home Economics (Appendix 1) and by the University of Alberta Hospitals Board Special Services and Research Committee, Faculty of Medicine, University of Alberta, Edmonton, AB (Appendix 2).

SUBJECT SELECTION

Ten non-insulin-dependent diabetic (NIDDM) male subjects took part in the study. Criteria used to select subjects were:

- free-living non-insulin-dependent-diabetic (Type II) males, age 20 - 60 yrs
- diabetes controlled by diet or diet/oral hypoglycemic agents (sulfonylureas only)
- body mass index (BMI) <35kg/m^2
- glycosylated hemoglobin (HbA_{1c}) <10%
- serum cholesterol <7mmol/L; serum triglycerides <5mmol/L
- no renal, hepatic, gastrointestinal or thyroid disease or complicating condition causing undue diet modification
- no clinical evidence of autonomic neuropathy
- not on lipid-lowering medication
- psychologically stable
- able to communicate in English
- resident of the greater Edmonton area for at least 6 months

prior to the study; available for regular follow-up

- eight or more starch choices/day in diet plan

Subjects were recruited through the University of Alberta Hospitals Metabolic Day Care, Edmonton, AB and the Misericordia Hospital Metabolic Clinic, Edmonton, AB. Of the eighty potential study participants contacted, (Appendix 3), thirteen individuals were willing to participate; eleven met all criteria and signed the consent form (Appendix 4). A subject information questionnaire (Appendix 5) was also completed by each participant. One subject was later disqualified. Ten subjects completed the study.

STUDY DESIGN

The 24 week study used a crossover experimental design with two periods (12 wks each). Five randomly chosen subjects ate the oat bran concentrate (OBC) bread first; the remainder ate the control white bread (WB) first. Individualized diet plans were formulated to provide: approximately 55% of total kcal as carbohydrate (CHO), 30% as fat and 15% as protein. Each diet plan incorporated a minimum of 8 servings per day of yeast bread/bread products as starch exchanges in the diet. During the OBC period, each subject used Quaker Oat Bran as his main cereal at breakfast. Cooked oatmeal was suggested as an alternative. During the WB period subjects avoided all oat products.

During the study, Dr. Ellen Toth provided medical management for each subject. The dietary intake of the ten subjects was monitored closely by the researcher. Patients were seen at three week intervals for dietary assessments, anthropometric measurements, and to discuss adherence to the diet and any questions/problems arising. The importance of maintaining body weight was emphasized. Throughout the study subjects continued normal daily activities and exercise patterns. Between interviews subject contact was maintained by telephone. Dietary counselling was provided as required. A 2 - 3 wk supply of frozen bread products was given to each subject and a record of the yeast bread/bread goods consumed was kept by the researcher. The study protocol is shown in Table 1.

DIETARY ASSESSMENT

A total of 18 days of dietary intakes were assessed quantatively for each subject by the recall method. Study dietary data were obtained as follows: study entry for 2 days, period 1 for 8 days, period 2 for 8 days. Recalls for period 1 and 2 included at least one weekend day but not more than 2 weekend days. Each subject was asked to recall, in chronological order, all foods and beverages consumed over the previous 48-hour period starting with the previous day and followed by the day prior to that. Food items were recorded on a dietary intake form (Appendix 6). Skilled probing by the

Table 1. Study protocol

Time	Study protocol
Wk 0	Subject's meal plan modified to incorporate at least 8 slices of bread. Day profile: Study entry. Two wk bread supply given to subject
Wk 2-10	Two wk supply of bread products provided. Dietary compliance evaluated
Wk 3-12	Dietary assessment via 48-hr recall at wks 3, 6, 9 and 12
Wk 12	Day profile: Period 1. Second period begins. Subject switched to other yeast bread/bread products. Two wk bread supply provided
Wk 14-22	Two wk supply of bread products provided. Dietary compliance evaluated
Wk 15-24	Dietary assessment via 48-hr recall at wks 15, 18, 21 and 24
Wk 24	Day profile: Period 2. Presentation of honorarium

researcher helped to ensure completeness of assessment. To estimate serving size, food models constructed according to Nutrition Canada (Health and Welfare Canada, 1973) specifications were used.

The daily nutrient intake of each subject was determined by coding all recorded food items using standardized coding procedures. Nutrient intakes were calculated by computer using a program based upon the Canadian Nutrient File (Health and Welfare Canada, 1985) plus data obtained from USDA Handbook #8 (Watt and Merrill, 1975; United States Department of Agriculture, 1976-1993). Values for dietary fibre and cholesterol were added to the nutrient data base from Southgate's tables (Paul and Southgate, 1978). Nutrient intakes for each day were calculated for the following nutrients: kilocalories (kcal), carbohydrate, sugar, starch, dietary fibre, crude fibre, protein, fat, cholesterol, saturated fat, monounsaturated fat, polyunsaturated fat and several vitamins and inorganic elements. In addition, mean daily intakes of food groups and contribution of food groups to total carbohydrate were obtained (Davenport, 1964).

ANTHROPOMETRIC DATA

At study entry, wks 12 and 24, the following anthropometric measurements were obtained: weight, height, relative body weight (Metropolitan Life Insurance Co., 1959), upper mid-arm circumference (MAC), triceps skinfold thickness

(TSF) using Lange skinfold callipers and mid-arm muscle circumference (MAMC).

BREAD AND BREAD PRODUCTS

Yeast breads, buns and muffins for use in this clinical study were developed in the Department of Food Science and Nutrition, University of Alberta. The high fibre bread, buns and muffins contained 45% oat bran concentrate. The oat bran concentrate, supplied by Dr. Y. Malkki, Exavena Oy/Inc, Finland, contained 45% total dietary fibre and 22.8% soluble dietary fibre as ß-glucan (Malkki, 1992a). Three similar batches of oat bran concentrate were used. Particle size of the oat bran concentrate was determined (six replications) by sieving analysis (Donelson and Yamazaki, 1972) using a nest of six 8-inch sieves with openings of 425 μm, 250 μm, 180 μm, 150 μm, 125 μm and 106 μm. Most of the oat bran concentrate particles (98.7 - 98.2%) passed through sieves with openings ≥180 μm. Using the formulations and methods developed in our laboratory, the Bakery Division, Northern Alberta Institute of Technology, Edmonton, made the yeast bread/buns (Table 2), and the Patient Support Centre, University of Alberta Hospitals, Edmonton, the muffins (Table 3). Bread and buns were baked in two lots, muffins (plain, raisin and cranberry) were made in three lots. Products were frozen (-29°C) and held until needed for distribution to the subjects. Oat bran cereal for the study was donated by the Quaker Oats Company (Quaker Oat Co

Table 2. Formulation for the study breads

Ingredient	Control WB	OBC Bread
Flour, Hard Red Spring Wheat	100.0g	55.0g
OBC		45.0g
Yeast, Fermipan instant	1.00g	1.85g
Vital wheat gluten		12.0g
Non-fat dry milk	4.0g	4.0g
Sodium stearoyl-2-lactylate (SSL)	0.5g	0.5g
Malt flour, 6-row barley		0.3g
Salt	2.0g	2.0g
Sugar, granulated	5.0g	5.0g
Shortening (Crisco)	3.0g	3.0g
Potassium bromate	45 ppm	45 ppm
Water (22°C)	58.0g	140.5g
Total weight	173.5g	269.15g

Method:

Have all ingredients at 22°C; measure into mixing bowl.

Combine by mixing on speed 1 (Hobart mixer) for 1-2 minutes.

Let rest for 5 minutes.

Mix at speed 3 for 6-7 minutes. Dough temperature should be 25-27°C.

Proof dough for 40 minutes at 90-110°F (38°C).

Punch down.

Mold into pan.

Proof bread at 100°F (90-110°) for 28 minutes.

Bake bread for 25 minutes: 210°C for first 10 minutes, then reduce heat to 200°C.

Cool on rack before packaging.

Table 3. Formulation for the study muffins

Ingredient	Control	OBC
Flour, All purpose	150.0g	82.5g
OBC		67.5g
Sugar, granulated	40.0g	30.0g
Baking powder (Magic)	6.0g	6.3g
Salt	2.0g	1.75g
Cinnamon (raisin, plain)	0.3g	0.3g
Nutmeg (raisin, plain)	0.3g	0.3g
Raisins/cranberries	45.0g	45.0g
Vegetable oil	27.5g	24.0g
Egg, beaten	25.5g	25.5g
Milk, fluid skim	135.0g	146.3g
Water		16.3g
Total weight		
Plain	386.6g	400.75g
Raisin	431.6g	445.75g
Cranberry	431.0g	445.15g

Method:

In mixing bowl, mix dry ingredients together; add liquid all at once.

Mix until just moistened (~17-20 strokes). Add raisins or cranberries before the last few strokes.

Measure dough (#20 scoop) into lightly greased muffin tins.

Bake 16-18 minutes at 200°C.

Cool in pans ~5 minutes. Remove from pans to cool completely.

- PLAIN MUFFINS: Use spices; omit raisins and cranberries.
- RAISIN MUFFINS: Use spices and raisins.
- CRANBERRY MUFFINS: Use chopped cranberries; omit spices.

Canada, Peterborough, ON). The fibre content of the oat bran concentrate bread and bread products is shown in Table 4.

QUESTIONNAIRES

Each subject completed a questionnaire on the overall acceptability of the oat bran concentrate bread products (Appendix 7). At study termination each subject was given a second questionnaire (Appendix 8) regarding his well being during the study (at entry, periods 1 and 2).

BIOCHEMICAL ANALYSIS

The following biochemical data were obtained at study entry and wks 12 and 24: fasting blood glucose, glycosylated hemoglobin (Hb), total plasma cholesterol, HDL cholesterol and triglycerides. Day profiles (ie. 8-hour blood glucose/insulin profiles) were conducted at study entry (wk 0) and at the end of each dietary period (wks 12 and 24).

The blood glucose analyses were performed by the glucose oxidase method (Kaplan, 1987) using a YSI semi-automatic glucose analyzer (model 27, Yellow Springs Incorporated, Yellow Springs, OH, U.S.A.). All procedures followed University of Alberta biosafety workplace regulations regarding human body fluids (level II biohazardous substances) (Workplace Hazardous Materials Information System, 1989). Prior to analysis plasma samples were thawed for 20 to 30 min at 22°C and kept refrigerated until analysis. Each day

Table 4. Dietary fibre in the bread exchanges consumed by NIDDM[1] subjects

Bread Product	TDF[3]	SDF[4]
OBC[2] bread (1.3 cm slice)	3.1g	1.6g
OBC[1] bun	3.7g	1.9g
OBC[1] muffin	3.5g	1.8g
Quaker oat bran cereal (234 mL cooked)	4.2g	1.4g
Oatmeal cereal (234 mL cooked)	2.5g	0.8g

[1] Non-insulin-dependent diabetic
[2] Oat Bran Concentrate
[3] Total dietary fibre
[4] Soluble dietary fibre

standards were run prior to sample analysis. A 25 μL Syringepet™ was used to inject sample into the glucose analyzer. Thirty sets of 21 blood plasma samples were analyzed in random order; samples within each set were randomized prior to analysis. The glucose concentration value for that sample was the average of three readings. Readings were converted from mg/dL to mmol/L using the conversion factor 0.05551 (SI Manual, 1985).

Area under the glucose and insulin curves were calculated by the method of Wolever and Jenkins (Wolever and Jenkins, 1986) using the formula:

$$\text{Area} = (A+B+C+D/2)t + DD^2t/2(D+E)$$

where A, B, C, D and E = positive glucose/insulin increments and t = time intervals between the samples. Fasting value was used as the baseline; areas beneath fasting values were ignored.

Glucose and insulin excursions were calculated as the difference between each peak and the lowest glucose/insulin concentration during the 8-hour curve (Braaten et al., 1991). The glycemic response area was divided into two 4-hour periods to evaluate separately the response to the breakfast meal (peak #1) and the lunch meal (peak #2). Insulin/glucose ratios were expressed as the ratio of peak serum insulin value over peak plasma glucose value (Morgan et al., 1979).

Glycosylated Hb was determined on samples of whole blood by high performance liquid chromatography (BioRad Diamat) at

the Department of Laboratory Medicine, University of Alberta Hospitals. Insulin assays were performed by the Muttart Diabetic Research and Training Centre using Pharmacia Insulin RIA 100 radioimmunoassay kits (Kabi Pharmacia Diagnostics AB, Uppsala, Sweden, 1993).

Total cholesterol, HDL cholesterol and triglyceride in the stored plasma samples were determined using Sigma Diagnostic enzymatic kits. LDL cholesterol was calculated using the formula: LDL-C = Total C - (HDL-C + TG/5) (Friedewald et al., 1972). Values were converted from mg/dL to mmol/L using the conversion factor 0.02586 for cholesterol and 0.01129 for triglyceride (SI Manual, 1985). Analyses were by Dr. M. Garg, University of Newcastle, New South Wales, Australia.

8-HOUR DAY PROFILE

For the day glucose profiles, subjects were admitted as outpatients to the Clinical Investigation Unit, University of Alberta Hospitals after an overnight fast. An intravenous catheter with normal saline was inserted into the patient's arm to allow multiple blood sampling. Blood samples for glucose and insulin analysis were drawn at time 0 and at 30 min intervals for eight hrs, except for one hr following main meals, when samples were taken at 15 min intervals. Twenty-one samples were collected on each profile day for plasma glucose and insulin analysis.

Breakfast, lunch and snacks typical of each subject's usual intake were provided. Bread products were representative of the study period just completed. Breakfast was served immediately following the fasting blood sample at 0 hrs (0 min), a snack at 2 hrs (120 min), lunch at 4 hrs (240 min) and a snack at 6 hrs (360 min). Quiet activity on the hospital unit was permitted. Each subject was discharged following collection of the last sample.

Samples for lipid, glucose and insulin analysis were centrifuged, frozen (-4°C for 24 hrs) and stored at -26°C for later chemical analysis.

DATA ANALYSIS

Dietary, biochemical [including maximum and minimum plasma glucose and serum insulin values and areas under the incremental glucose/insulin curve (AUC)] and clinical data for the WB and OBC dietary periods were subjected to analyses of variance (ANOVA) using SAS GLM (SAS, 1985). Sources of variation for the crossover design (Petersen, 1985) were group (g=2), subjects within group (s=4), time (t=2) and dietary periods (dp=2).

Differences between the pre-dietary period and 2 study dietary periods for mean plasma and insulin eight-hour profile values, measured 21 times for each subject, were compared using repeated measures analysis of variance.

Correlations between dietary and metabolic variables were computed within dietary period by subject using repeated measures analyses of variance. Sources of variation were the same as for the crossover analyses of variance. Correlations between change in total cholesterol values (total cholesterol for WB minus total cholesterol for OBC) and the amount of soluble fibre consumed in the OBC bread were computed using pairs of the 2 values for each person.

Differences between the pre-dietary period and study periods for dietary, clinical and biochemical data were compared using ANOVA (SAS GLM). Sources of variation were group (g=2), subjects within groups (s=4), study (st=2) and group*study.

The effects of oral sulfonylureas on metabolic variables were determined using analyses of variance. Sources of variation were group (g=2), drug (d=2), group*drug, subjects within group*drug (S_{ij}=3,1,2,2), time (t=2), dietary period (dp=2) and drug*dietary period. Source of error for drug was subjects within group*drug.

RESULTS

There were 10 subjects in the study. For this thesis, data for 8 subjects were statistically analyzed. Two of the 10 participants had HbA_{1C} values >10% at study entry and thus did not meet entry criteria; results from the study entry profile were not available until several weeks after the study began. The two subjects were kept in the study; their results are included in Appendix 9.

STUDY ENTRY

SUBJECT CHARACTERISTICS

Subject profile information for the male NIDDM subjects at study entry is shown in Table 5. The mean age of the participants was 45.5 ± 0.51 yrs. Mean body weight was 82.9 ± 0.34 kg. Mean body mass index (BMI) was 27.6 ± 0.15 kg/m^2 and average relative body weight (Metropolitan Life Insurance Co., 1959) was 126.5 ± 0.49 %. At study entry the mean HbA_{1C} value of subjects was 6.94 ± 0.31 % and the fasting plasma glucose was 8.21 ± 0.41 mmol/L. Study entry mean fasting serum insulin was 13.50 ± 2.18 μU/L. Subject mean plasma lipid values at entry were 4.65 mmol/L for total cholesterol and 2.26 mmol/L for triglyceride at over the 10th and 75th percentile, respectively (Gotto et al., 1984).

Table 5. Characteristics of NIDDM[1] subjects

Characteristic	Mean ± SEM[2]	Range
Age (yrs)	45.5 ± 0.51	39 - 57
Height (cm)	174 ± 0.58	165 - 190
Weight (kg)	82.9 ± 0.34	64.2 - 95.5
BMI[3] (kg/m^2)	27.6 ± 0.15	22.2 - 33.8
RBW[4] (%)	126.5 ± 0.49	100.0 - 153.0
HbA_{1C}[5] (%)	6.94 ± 0.31	5.2 - 10.2
Plasma Glucose[6] (mmol/L)	8.21 ± 0.41	5.77 - 12.51
Serum Insulin[6] (μU/L)	13.50 ± 2.18	4.0 - 32.0
Plasma Cholesterol[6] (mmol/L)	4.65 ± 0.12	3.10 - 7.30
Plasma TG[6,7] (mmol/L)	2.26 ± 0.24	0.60 - 4.40

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Body mass index
[4] Relative body weight
[5] Glycosylated hemoglobin
[6] Fasting values
[7] Triglyceride

Mean duration of diabetes for subjects (Table 6) was 2.1 years. Five subjects controlled their diabetes with diet only, 3 with diet plus oral sulfonylureas. Two subjects took medication to control high blood pressure and 2 took vitamin supplements regularly. All participants were married; 5 participated in the daily meal preparation and grocery shopping either solely or with their spouse.

DIETARY INTAKE

The mean daily nutrient intake of NIDDM subjects at study entry is given in Table 7. At entry the mean carbohydrate intake of subjects was 48% of energy intake, protein was 19% and fat was 31%. Total dietary fibre intake was 17 g/d or 8.5 g/1000 kcal. Cholesterol intake of subjects was 272 mg/d and mean saturated fatty acid intake was 9.8% of total energy intake. Average P/S ratio of the subjects' diet was 0.7.

THE STUDY

DIETARY INTAKE

The mean macronutrient composition of the subject's diets during each of the two study periods is shown in Table 8. There were no significant differences in total energy, percent carbohydrate, protein or fat, or intake of carbohydrate or fat (g/d) intake between the WB and OBC periods. During the OBC

Table 6. Subject profile information

Subject	Age (yr)	Duration of Diabetes (yrs)	Diabetic Therapy	Vitamin/ Mineral Supplement	Diet Habits: Shopping/ Cooking
001	41	1	Diet	No	wife/wife
003	43	1	Diet	No	self/self
004	44	1	Diet + SU[1]	No	wife/wife
005	52	4	Diet + SU	Multi-vitamin	both[2]/both
006	47	3	Diet	No	both/both
007	39	2	Diet + SU	Calcium	self/self
008	57	4	Diet	Vitamin E	wife/wife
010	41	1	Diet	No	both/both

[1]Sulfonylurea drugs
[2]Subject and wife

Table 7. Mean daily nutrient intake of NIDDM[1] subjects at study entry

Variable	Mean ± SEM[2]	Range
Energy		
(kcal/d)	2012 ± 126	890 - 2889
Carbohydrate		
total (g/d)	241.5 ± 20.1	112.5 - 366.5
% of kcal	50.0 ± 1.8	42.1 - 58.6
dietary fibre (g/d)	17.0 ± 2.6	1.1 - 40.1
starch (g/d)	159.3 ± 12.1	93.9 - 263.5
sugar (g/d)	67.4 ± 2.8	2.0 - 138.4
sugar/starch ratio	0.33 ± 0.03	0.02 - 0.66
Protein		
total (g/d)	92.9 ± 4.9	41.6 - 123.8
% of kcal	19.0 ± 0.6	17.2 - 21.8
Fat		
total (g/d)	67.5 ± 6.5	52.0 - 89.2
% of kcal	31.0 ± 1.9	23.5 - 39.6
SFA[3] (g/d)	21.2 ± 1.8	9.9 - 31.5
P/S[4] ratio	0.7 ± 0.1	0.5 - 1.1
cholesterol (mg/d)	271.8 ± 41.8	122.0 - 568.0

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Saturated fatty acid
[4] Polyunsaturated/saturated fatty acid

Table 8. Mean daily nutrient intake of NIDDM[1] subjects

Variable	WB Period	OBC Period
	Mean ± SEM[2]	
Energy (kcal/d)	2411 ± 79	2567 ± 79
Carbohydrate (g/d)	301.8 ± 11.6	336.9 ± 11.6
(% total kcal)	50.9 ± 1.0	52.8 ± 1.0
starch	206.2 ± 12.1	225.2 ± 12.1
sugar	86.5 ± 2.8	83.4 ± 2.8
sugar/starch ratio	0.33 ± 0.02	0.35 ± 0.02
Protein (g/d)	103.7 ± 2.8	116.0 ± 2.8*
(% total kcal)	17.6 ± 0.4	18.6 ± 0.4
Fat (g/d)	85.2 ± 4.4	81.7 ± 4.4
(% total kcal)	31.5 ± 1.2	28.6 ± 1.2
Cholesterol (mg/d)	343.8 ± 43.1	280.6 ± 43.1
Saturated Fat (g/d)	27.4 ± 2.1	24.6 ± 2.1
(% of total kcal)	10.0 ± 0.6	8.6 ± 0.6
Monounsaturated Fat (g/d)	32.2 ± 1.4	31.0 ± 1.4
(% of total kcal)	11.8 ± 0.4	10.7 ± 0.4
Polyunsaturated Fat (g/d)	19.3 ± 0.9	18.4 ± 0.9
(% of total kcal)	7.1 ± 0.3	6.4 ± 0.3
P/S[3] Ratio	0.7 ± 0.04	0.8 ± 0.04

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Polyunsaturated/saturated fatty acid
* Significant at $p \leq 0.05$

period subject protein intake (g/d) was higher ($p \leq 0.05$) than in the WB period. However, variation in protein intake among individual subjects was considerable. Dietary fat intake of subjects did not differ in either quantity or quality.

Subjects were asked to consume at least 8 slices bread/bread products/d throughout the study. The mean bread/bread products consumption of subjects was 5.9 ± 0.6 bread choices/d in the WB period and 5.7 ± 0.6 in the OBC period (NS). One subject ate 9.5 slices/d in the OBC period. Three subjects were unable to eat more than 5 slices/d in the OBC period because of flatulence. Two subjects had difficulty consuming the required amount of bread/bread products because of ethnic food practices.

The food consumption pattern of NIDDM men during the study periods, expressed as mean daily intake of food groups, is presented in Table 9. The total mean daily intake of cereals during the OBC period was higher than during the WB period (NS) and as expected, mean whole grain cereal consumption was higher ($p \leq 0.001$) in the OBC period than in the WB period. In the OBC period intake of meat was lower than in the WB period; mean daily intake (g) of total meat (MPFE) group ($p \leq 0.05$) and the meat subgroup ($p \leq 0.001$) were significantly lower. Intake of the high fibre vegetable subgroup was also significantly lower ($p \leq 0.05$) for the OBC period than the WB period. Throughout the 2 dietary periods,

Table 9. Mean daily intake (g) of food groups by subjects

Food Group	WB Period	OBC Period
	Mean ± SEM[1]	
Cereals		
Total	457.2 ± 39.5	553.4 ± 39.5
Refined	432.8 ± 37.8	481.2 ± 39.9[2]
Whole Grain	11.9 ± 4.9	81.8 ± 5.0***
Vegetables		
Total	319.6 ± 27.1	289.0 ± 27.1
High Vitamin C	22.5 ± 7.4	42.9 ± 7.6
High Carotene	90.5 ± 10.5	82.1 ± 10.8
Legumes	31.1 ± 23.4	46.4 ± 24.1
High Fibre	18.3 ± 1.4	10.9 ± 1.4*
Other	173.1 ± 22.7	100.9 ± 23.4
Fruits		
Total	209.4 ± 21.2	208.6 ± 21.2
Citrus	12.7 ± 9.9	10.0 ± 10.2
Other	113.4 ± 26.4	150.9 ± 27.2
Juice	85.6 ± 10.3	59.6 ± 10.6
Fats and Oils		
Total	32.8 ± 2.5	31.2 ± 2.5
Butter	2.0 ± 1.3	0.1 ± 1.3
Margarine	15.9 ± 3.6	15.3 ± 3.7
Oils	4.6 ± 1.3	5.5 ± 1.3
Other	8.1 ± 1.0	8.7 ± 1.0
Dairy		
Total	331.2 ± 27.4	388.5 ± 27.4
Milk	338.5 ± 28.1	386.9 ± 28.9
Cheese	9.0 ± 5.4	24.7 ± 5.6
Desserts	14.0 ± 3.9	5.0 ± 4.0
MPFE[3]		
Total	206.2 ± 9.9	159.1 ± 9.9*
Meat	129.2 ± 3.4	72.2 ± 3.5***
Poultry	33.9 ± 5.0	47.0 ± 5.1
Fish	25.7 ± 9.3	31.9 ± 9.6
Eggs	25.5 ± 7.6	15.4 ± 7.8
Nuts	1.8 ± 2.2	5.1 ± 2.2
Foods primarily sugar	16.8 ± 5.2	6.0 ± 5.2
Miscellaneous		
Total[4]	179.4 ± 32.8	152.3 ± 32.8
Soups	140.6 ± 34.6	107.2 ± 35.7
Mixtures	11.1 ± 4.1	5.6 ± 4.2
Alcohol	32.4 ± 18.9	42.0 ± 19.5
Total[3] (g/d)	1754.3 ± 45.2	1793.2 ± 45.2

[1] Mean ± standard error of the mean
[2] Unequal observations account for differences in SEM
[3] Meat, Poultry, Fish and Eggs
[4] Does not include soft drinks, tea, coffee or alcohol
*,**,*** Significant at $p \leq 0.05$, $p \leq 0.01$ and $p \leq 0.001$, respectively

food intake of the subjects did not change appreciably except for bread intake.

The total carbohydrate intake of subjects was similar in both dietary periods. Table 10 shows the contribution of the cereal group to the intake of total carbohydrate, starch and dietary fibre. During the OBC period, the cereal food group including bread contributed significantly more ($p \leq 0.05$) total carbohydrate, starch and dietary fibre to the diet than during the WB period.

The mean fibre content of the diets is given in Table 11. As expected, in the OBC period the intake of total dietary fibre (TDF) was significantly higher than in the WB period ($p \leq 0.01$). For the OBC period, subjects consumed an average of 40 g/d of oat bran concentrate; oat bran concentrate provided 52% of the total dietary fibre intake. Twenty-three percent of the oat bran concentrate is soluble fibre.

ANTHROPOMETRICS

Anthropometric measurements of NIDDM subjects (Table 12) did not change significantly over the 24 wk study period. Mean body weight, BMI and relative body weight of subjects were similar during the 2 dietary periods. In addition, arm circumference, triceps skinfold thickness, calculated mid arm muscle circumference (MAMC) and % standard MAMC of subjects were the same during both periods of the study. The average

Table 10. Proportion of carbohydrate components contributed by cereal group to the total intake of carbohydrate, starch and dietary fibre

	Total CHO[1]		Starch		Dietary Fibre	
	g	%	g	%	g	%
Cereal Food Group Intake (/d)						
WB Period	179	60	169	82	8.0	43
OBC Period	225	67*	200	89*	23.6	70*
Total CHO[1] Intake (/d)						
WB Period	298	100	206	100	18.5	100
OBC Period	336	100	225	100	33.7**	100

[1] Carbohydrate
*,** Significant at $p \leq 0.05$ and $p \leq 0.01$ respectively

Table 11. Mean fibre content of diets of NIDDM[1] subjects

Variable	WB Period	OBC Period
	Mean ± SEM[2]	
Total Dietary Fibre (g/d)	18.5 ± 2.2	33.7 ± 2.2**
Total Dietary Fibre from OBC (g/d)	0.00	17.4 ± 1.2***
Soluble Dietary Fibre from OBC (g/d)	0.00	8.6 ± 0.6***

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
, * Significant at $p \leq 0.01$, $p \leq 0.001$, respectively

Table 12. Mean anthropometric measurements of NIDDM[1] subjects

Variable	WB Period	OBC Period
	Mean ± SEM[2]	
Weight (kg)	82.93 ± 0.47	82.94 ± 0.41[3]
BMI[4] (kg/m^2)	27.50 ± 0.22	27.56 ± 0.19
RBW[5] (%)	125.60 ± 0.66	125.43 ± 0.57
Arm Circumference (cm)	32.43 ± 0.00	32.43 ± 0.00
TSF[5] (mm)	14.48 ± 0.16	14.41 ± 0.16
MAMC[7] (cm)	27.86 ± 0.05	27.89 ± 0.05
% Standard MAMC[7]	101.56 ± 0.20	101.60 ± 0.20

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Unequal observations account for differences in SEM
[4] Body mass index
[5] Relative body weight
[6] Triceps skinfold thickness
[7] Mid arm muscle circumference

relative body weight, was similar throughout both study periods.

BIOCHEMICAL ASSESSMENT

Table 13 summarizes blood glucose and insulin data for the 2 study periods. Mean glycosylated hemoglobin levels, fasting plasma glucose values and serum insulin values were similar for the 2 periods. Figures I and II show the mean plasma glucose and serum insulin profiles for both study periods (WB period and OBC period). Incremental areas under the glucose and insulin curves (AUC) were 40.9% and 16.8%, respectively, lower ($p \leq 0.05$ and NS) in the OBC period than in the WB period (Table 13). During the OBC period, all subjects (except one, whose area under the curve remained virtually the same) experienced a flattening of the total glucose curve as expressed by area under the curve. During the OBC period mean area under the curve following the first meal (breakfast - peak #1) for glucose and insulin was 34.7% and 21.5%, respectively, lower ($p \leq 0.01$ and $p \leq 0.05$) and following the second meal (lunch - peak #2), 50.2% and 12.9%, respectively, lower (NS) than in the WB period. Mean plasma glucose and serum insulin values for all times in the profile (Figures I and II, respectively) were lower (NS) for the OBC period than the WB period, except at 15 and 30 min for glucose and 30 and

Table 13. Blood glucose and insulin parameters for NIDDM[1] subjects

Variable	WB Period	OBC Period
	Mean ± SEM[2]	
HbA_{1c} (%)	8.14 ± 0.33	7.71 ± 0.33
Fasting levels		
Plasma Glucose (mmol/L)	8.86 ± 0.47	8.68 ± 0.47
Serum Insulin (μU/L)	14.1 ± 2.44	16.6 ± 2.44
Glycemic response area		
• Glucose (mmol·min/L)		
total area (8hr)	949 ± 98	561 ± 98*
part 1 area (4hr)	568 ± 25	371 ± 25**
part 2 area (4hr)	381 ± 86	190 ± 86
• Insulin (μU·min/L)		
total area (8hr)	11560 ± 685	9614 ± 685
part 1 area (4hr)	5268 ± 301	4136 ± 301*
part 2 area (4hr)	6291 ± 483	5478 ± 483
Peak #1 response		
• Glucose (mmol/L)		
maximum	15.4 ± 0.8	13.4 ± 0.8
excursion	6.63 ± 0.46	5.62 ± 0.46
• Insulin (μU/L)		
maximum	73.5 ± 2.2	62.3 ± 2.2*
excursion	43.5 ± 4.5	38.6 ± 4.5
Peak #2 response		
• Glucose (mmol/L)		
maximum	13.1 ± 1.0	11.1 ± 1.0
excursion	4.43 ± 0.64	3.34 ± 0.64
• Insulin (μU/L)		
maximum	76.4 ± 2.5	68.0 ± 2.5
excursion	34.0 ± 6.0	36.8 ± 6.0
Insulin/glucose ratio		
• Peak 1	5.20 ± 0.23	4.93 ± 0.23
• Peak 2	6.33 ± 0.57	6.24 ± 0.57

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
*,** Significant at $p \leq 0.05$, $p \leq 0.01$, respectively

Figure I. Mean 8-hour Plasma Glucose Profile

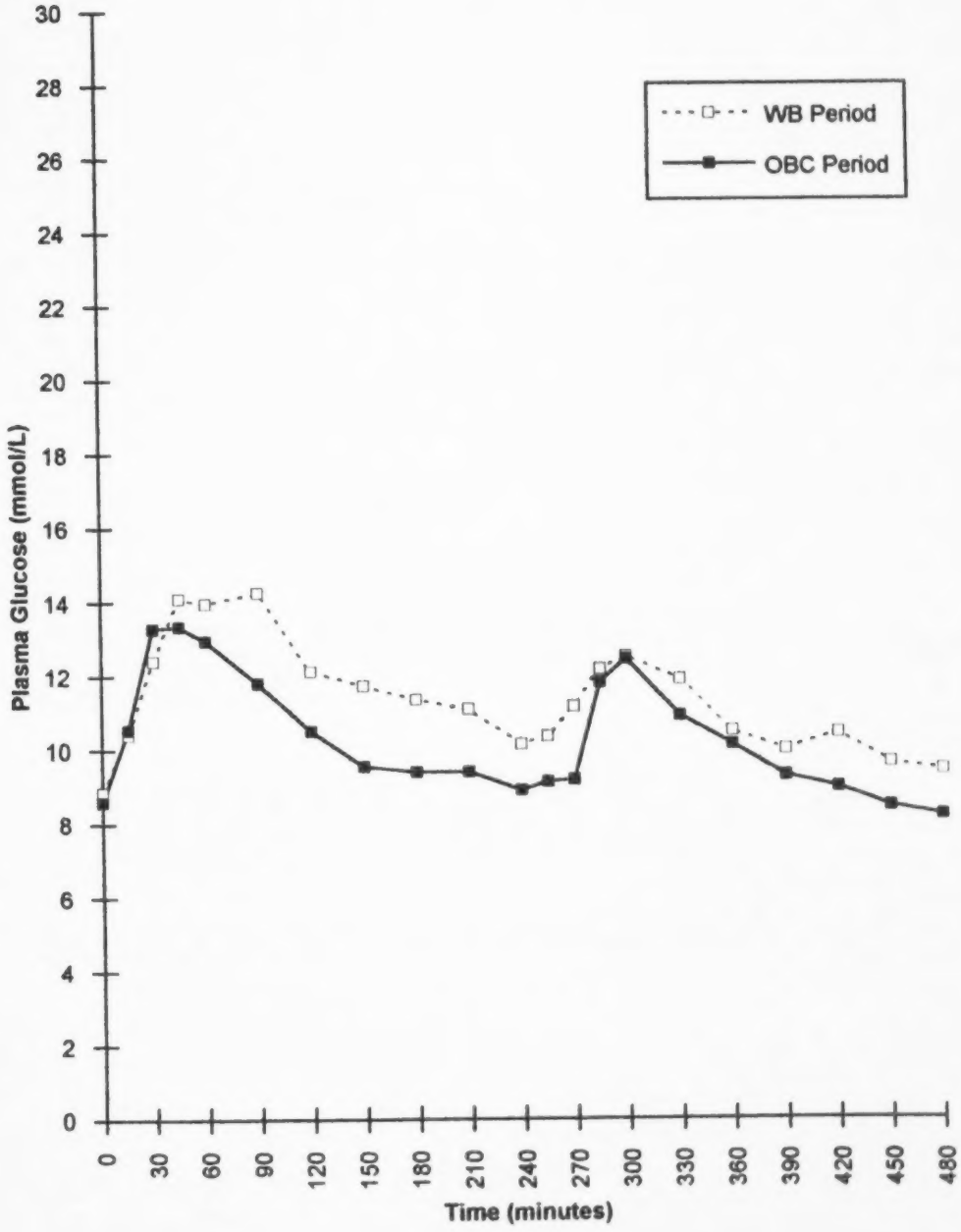
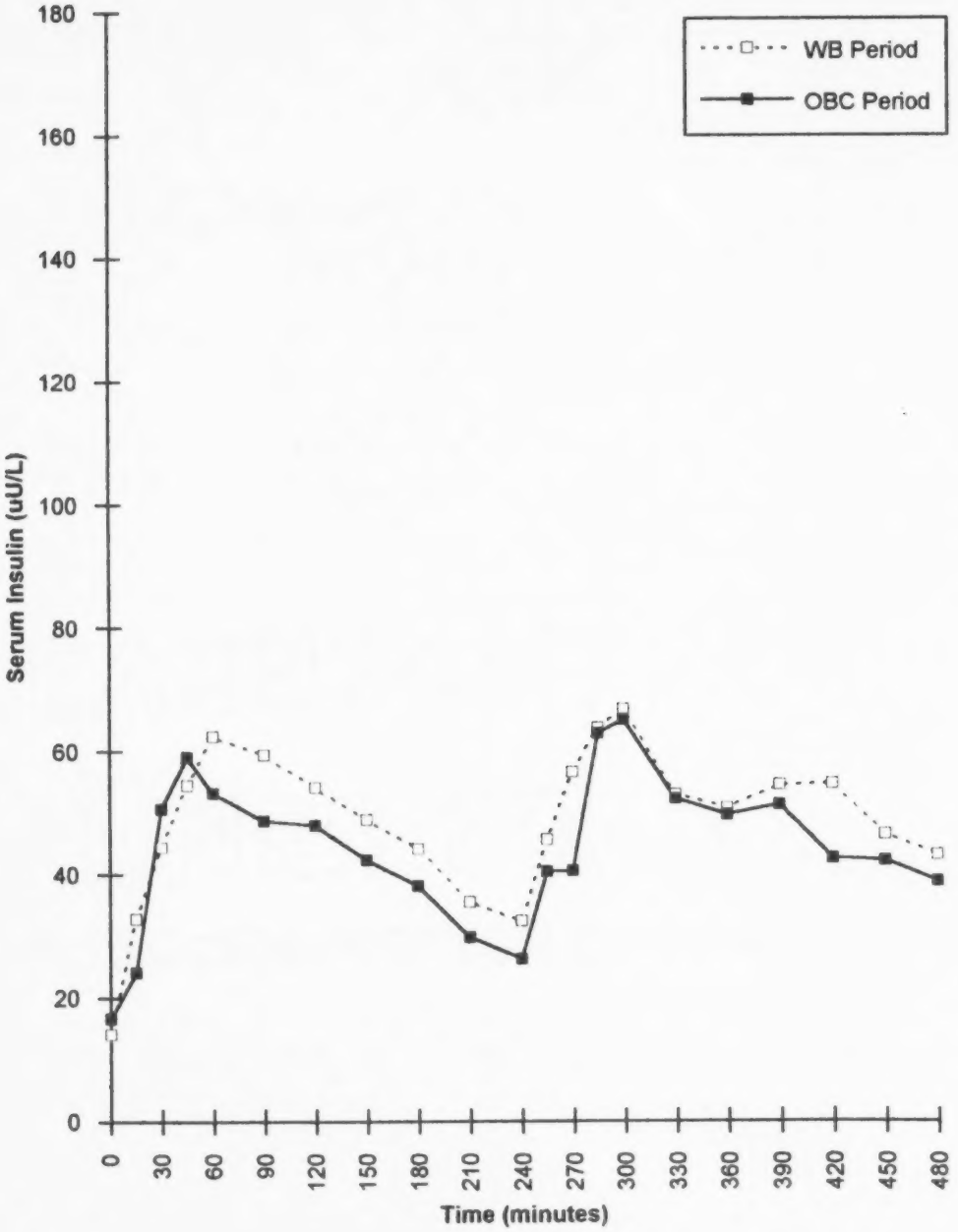


Figure II. Mean 8-Hour Serum Insulin Profile



45 min for insulin when values for the OBC period were slightly higher than those of the WB period.

The mean maximum plasma glucose values for peaks 1 and 2 were 13.2% and 15%, respectively, lower (NS) in the OBC period than in the WB period. In the OBC period, mean serum insulin values for peaks 1 and 2 were 15.3% and 11.0%, respectively, lower ($p \leq 0.05$ and NS) than in the WB period. Mean minimum serum insulin values for subjects were similar in the study periods. Mean times for maximum serum insulin values following the first and second meals were 26 min and 30 min, respectively, earlier ($p \leq 0.05$ and NS) in the OBC period than in the WB period.

In the OBC phase, the mean glucose response (excursion) of subjects was slightly lower (NS) than in the WB period (Table 13). The mean peak difference of fasting plasma glucose from maximum (peak) values for subjects after the first and second meals in the OBC period was 28.2% and 35%, respectively, lower than after the WB period (data not shown). Compared to the WB period, glucose excursions for NIDDM subjects were reduced by 13.7% after the first meal and 24.6% following the second meal in the OBC period.

The mean insulin response (excursion) of subjects in the OBC period was lower for peak 1 (NS) and slightly higher for peak 2 than in the WB period (Table 13). Insulin/glucose ratio (Table 13) for the 2 dietary periods were similar, however, in

the OBC period, there was a trend (NS) toward a lower insulin/glucose ratio.

No significant correlations were determined between dietary variables and glucose parameters for subjects. Significant negative correlations ($p \leq 0.05$) were found between total dietary fibre, total dietary fibre from oat bran concentrate, soluble fibre from oat bran concentrate and total insulin area under the curve (Table 14).

Mean plasma lipid values for NIDDM subjects in the study periods are given in Table 15. Compared to the WB period, mean plasma total-cholesterol (C) and LDL-C of subjects were lower ($p \leq 0.01$) by 0.74 mmol/L and 0.77 mmol/L (14% and 23%), respectively, in the OBC period. The mean HDL-C of subjects was similar in both periods. Total-C/HDL-C of subjects was decreased by 14% while on the OBC diet. LDL/HDL ratio was 25% lower ($p \leq 0.05$) in the OBC period than in the WB period. Although plasma triglyceride (TG) was lower by 0.11 mmol/L (5%) in the OBC period than in the WB period, the difference was non significant. Mean plasma triglyceride at study entry was decreased by 10% at the end of both study periods.

Subjects consuming the greatest amount of soluble fibre from oat bran concentrate appeared to show the greatest reduction in plasma cholesterol levels between the 2 diet periods (Table 16); however this was not significant. There was no effect of sulfonylurea/diet treatment or diet sequence

Table 14. Relationships between dietary and biochemical parameters of NIDDM[1] subjects

Dietary Variable	Biochemical Variable	Correlation Value	Level of Significance
Total dietary fibre (TDF)	Total AUC (insulin)	-0.75	$p \leq 0.050$
TDF from OBC	Total AUC (insulin)	-0.79	$p \leq 0.034$
SF from OBC	Total AUC (insulin)	-0.79	$p \leq 0.033$

[1] Non-insulin-dependent diabetic

Table 15. Mean plasma lipid values for NIDDM[1] subjects

Variable	WB Period	OBC Period
	Mean ± SEM[2]	
Total Cholesterol (mmol/L)	5.3 ± 0.11	4.6 ± 0.11**
LDL[3] Cholesterol (mmol/L)	3.36 ± 0.12	2.59 ± 0.12**
HDL[4] Cholesterol (mmol/L)	0.96 ± 0.05	1.04 ± 0.05
Triglycerides (mmol/L)	2.14 ± 0.16	2.03 ± 0.16
Total-C/HDL Ratio	5.4 ± 0.25	4.7 ± 0.25
LDL/HDL Ratio	3.54 ± 0.20	2.68 ± 0.20*

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Low density lipoprotein
[4] High density lipoprotein
*, ** Significant at $p \leq 0.05$ and $p \leq 0.01$, respectively

Table 16. Glucose and lipid reduction[1] as well as soluble fibre intake of individual NIDDM[2] subjects

Subject	Total SF	Area under curve Δ		Chol[3] Δ	LDL-C Δ
	g/d	Glucose mmol·min/L	Insulin μU·min/L	mmol/L	mmol/L
001	6.88	-715	+2542	-0.90	-0.99
003	14.20	-175	-5235	-1.00	-0.72
004	6.47	-138	-1383	-0.10	+0.02
005	9.30	-1096	-1256	-2.10	-2.27
006	8.91	-681	-4815	-0.30	-0.23
007	6.16	+4	-3794	-0.50	-0.24
008	7.54	-195	+25	-0.20	-0.20
010	8.91	-111	-1655	-0.80	-1.51
Mean Δ	8.55	-388	-1946	-0.73	-0.73
% Δ		-41	-17	-14	-23

[1] Difference between values in WB and OBC diet periods
[2] Non-insulin-dependent diabetics
[3] Plasma cholesterol

on biochemical variables. Subjects on sulfonylurea drugs did not reduce drug dosage.

CHANGE IN DIETARY PATTERNS

At study entry the mean total energy intake of NIDDM subjects was significantly lower than for each of the study periods, which did not differ (Table 17). Subject mean carbohydrate intake (g/d) in each study period was similar and significantly higher than that at study entry. Protein intake (g/d) of subjects in the OBC period was higher ($p \leq 0.05$) than at study entry; subject protein intake in the WB period was similar to that at study entry and the OBC period. There were no differences in mean fat intake (g/d) or in percentages of macronutrient intake at study entry and during the 2 dietary periods.

The mean food intake (g/d) from food groups by subjects was similar at study entry and in the 2 study periods (Table 18; Figure III). Mean cereal consumption (g/d) by subjects was higher in the OBC period than in the WB period (NS) and at study entry ($p \leq 0.05$). Although not significantly different, daily fruit intake by subjects was higher during the study than at study entry. Consumption of MPFE was significantly higher in the WB period than in the OBC period. At all times, subjects consumed low amounts of foods primarily sugar. The mean daily consumption of all other food groups was similar for all periods evaluated.

Table 17. Mean daily nutrient intake of NIDDM[1] subjects at study entry and during the 2 dietary periods

Nutrient	Study Entry	WB Period	OBC Period
	Mean ± SEM[2]		
Energy (kcal/d)	2010 ± 124[b]	2411 ± 124[a]	2657 ± 124[a]
CHO (g/d)	241.5 ± 20.1[b]	301.8 ± 20.1[a]	336.9 ± 20.1[a]
(% total kcal)	49.2 ± 1.8	50.9 ± 1.8	52.8 ± 1.8
Protein (g/d)	92.9 ± 4.9[b]	103.7 ± 4.9[ab]	116.0 ± 4.9[a]
(% total kcal)	19.4 ± 0.6	17.6 ± 0.6	18.6 ± 0.6
Fat (g/d)	67.5 ± 6.5	85.2 ± 6.5	81.7 ± 6.5
(% total kcal)	31.4 ± 1.9	31.5 ± 1.9	28.6 ± 1.9

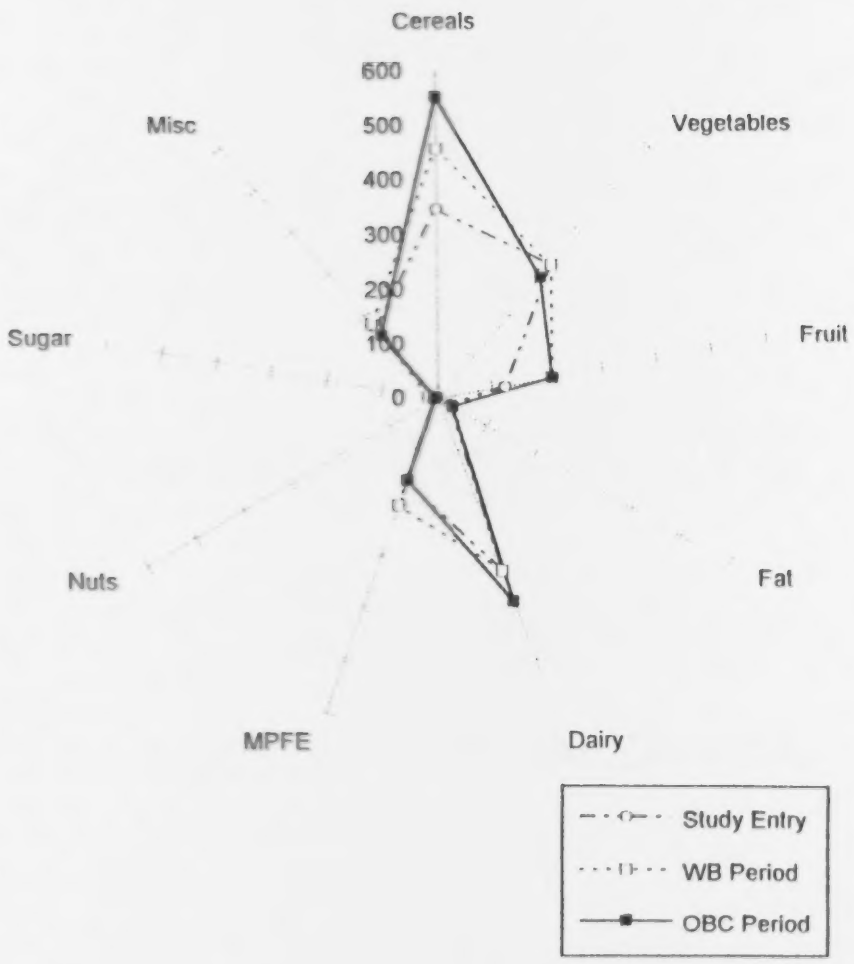
[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[ab] Means not followed by the same letter in each row are significantly different from each other at $p \leq 0.05$

Table 18. Mean daily intake (grams) of food groups at study entry and during the 2 dietary periods by NIDDM[1] subjects

Food Group	Study Entry	WB Period	OBC Period
		Mean ± SEM[2]	
Cereals	348 ± 54[b]	457 ± 54[ab]	553 ± 54[a]
Vegetables	320 ± 46	320 ± 46	289 ± 46
Fruits	123 ± 37	209 ± 37	209 ± 37
Fats and Oils	25 ± 5	33 ± 5	31 ± 5
Dairy	330 ± 37	331 ± 37	389 ± 37
MPFE[3]	161 ± 17[b]	206 ± 17[a]	159 ± 17[b]
Nuts	2 ± 2	2 ± 2	6 ± 2
Foods primarily sugar	1 ± 5	17 ± 5	6 ± 5
Miscellaneous[4]	168 ± 61	179 ± 61	152 ± 61
Total[4] (g/d)	1479 ± 119	1754 ± 119	1794 ± 119

[1] Non-insulin-dependent diabetic
[2] Mean ± standard error of the mean
[3] Meat, Poultry, Fish and Eggs
[4] Does not include soft drinks, tea, coffee or alcohol
[ab] Means not followed by the same letter in each row are significantly different from each other at $p \leq 0.05$

Figure III. Mean daily intake (g) of food groups at study entry and during the 2 dietary periods



QUESTIONNAIRES

Subject evaluations of the acceptability of the OBC bread products (Figure IV) showed that the OBC bread/bread products were excellent (overall score of 3.9 out of maximum of 5). Comments from the subjects regarding the oat bran concentrate bread products appear in Table 19. Seventy five percent of the subjects indicated a willingness to buy the products if commercially available.

Five of the 8 subjects answered the questionnaire regarding their feelings of well-being. Average overall well-being was: 11.6 before the study, 12.0 during the WB period and 9.0 during the OBC period (NS). Subjects' comments regarding well being are presented in Table 20. Side effects experienced by the subjects during the OBC phase of the study are shown in Table 21. Three subjects, who described their well-being to be slightly poorer in the OBC period than in the WB period, experienced flatulence. During the OBC period one subject felt extremely unwell due to the flu and a subsequent slow recovery. This subject was a thin Type II diabetic individual whose condition worsened progressively during the study period; his family physician was considering putting him on insulin. During the WB period, another subject had dental surgery. A third subject began shift work at study entry after day shifts for the previous 6 months. This same subject vacationed out of the country for 6 weeks during the WB

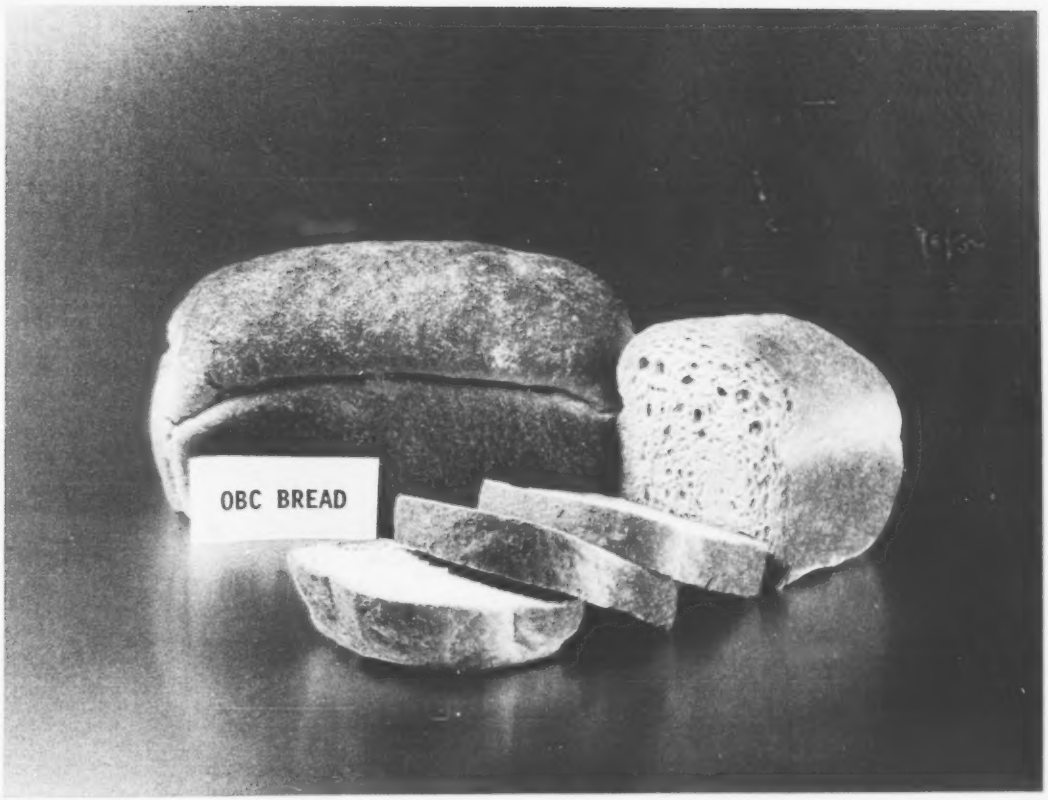


Figure IV. Oat bran concentrate bread

Table 19. Comments from NIDDM[1] subjects regarding the OBC bread products

Comment	Number of subjects[2]
•Liked the taste	4
•Preferred the OBC bread toasted	1
•OBC bread heavy	2
•Sour taste	1
•Liked moistness	1
•Buns dry	2
•Did not like the OBC muffins	2
•Liked the OBC muffins best	2
•Buns and muffins convenient to use	1
•OBC recipes need refinement	1
•Couldn't eat the OBC bread at all 3 meals/day	2
•Lack of variety in bread type monotonous	1
•Would buy if commercially available:	Yes (6) No (1) Maybe (1)
•Would eat OBC products even if disliked provided diabetic condition improved	1
•Other OBC products desired:	
Cookies	2
Cake	2
Raisin bread	1
Melba toast	1
Crackers	1

[1] Non-insulin-dependent diabetic
[2] No. of subjects = 8

Table 20. Comments from NIDDM[1] subjects regarding their own well-being

Characteristic	Before Study	WB Period	OBC Period
Tested blood sugars regularly			
Yes	4[2]	3	3
No	1	2	2
Sometimes	0	0	0
Blood sugars well controlled	5	5	4
Stable body weight	5	3	4
Weight loss	0	1	0
Weight gain	0	0	1
Gas (flatulence)	0	0	5
No energy	1	2	1
Followed diet plan			
Always	1	1	1
Usually	4	4	4
Sometimes	0	0	0

[1] Non-insulin-dependent diabetic
[2] Number of subjects (5 of 8 responded)

Table 21. Side effects experienced by NIDDM[1] subjects in the OBC[2] period of the study

Side effect	Number of subjects[3]
Feeling of fullness	6
Ate less at next meal	2
Longer time before eating next meal	1
Took longer to eat and digest	2
Could not finish sandwich	1
GI problems	6
Gas	5
Diarrhea (1st week)	1
Upset stomach	1

[1] Non-insulin-dependent diabetic
[2] Oat bran concentrate
[3] Number of subjects = 8

period. Another participant stated that he worked harder to control his blood sugar during the study. One requirement of the study was that weight remain constant; statistical analysis of NIDDM subjects' weight showed weight gain/loss during the study (data not presented) to be non significant.

DISCUSSION

SUBJECTS AND STUDY DESIGN

For the present 6-month study, the eight NIDDM diabetic subjects (males, ages 37 to 57 yrs) were living in their homes in Edmonton. The subjects had late onset diabetes, recently diagnosed ($\bar{x}$ = 2.1 yrs ago). For these subjects, the usual management of diabetes was diet therapy; three subjects required oral hypoglycemics (sulfonylureas). As is typical of NIDDM subjects, the study group was overweight with an average relative body weight of 127%; the range was 100 to 153%. At study entry, metabolic control of subjects was fairly good as indicated by the mean glycosylated hemoglobin value of 6.9%. The mean fasting plasma glucose value of 8.2 mmol/L, however, was rather high. Lipid profile data indicated hypertriglyceridemia at study entry; mean plasma triglyceride was 2.26 mmol/L (75th percentile), mean plasma total cholesterol was 4.65 mmol/L (10th percentile). This is a typical profile for non-insulin-dependent diabetic individuals (DeFronzo and Ferrannini, 1991; Coulston, 1994). At study entry, dietary assessments revealed that mean energy intake from carbohydrate, protein and fat was 50%, 19% and 31% of kcal. The level of fat intake was lower than that of the general population [for the typical North American a level of 36% to 42% of kcal has been reported (Grundy, 1991)]. Recently, for a US group of NIDDM subjects, a fat intake of

34% of kcal was reported (Schmidt et al., 1994). The mean dietary fibre intake at study entry was 17 g per day, a level of fibre intake similar to that reported for the typical North American male (13 g per day) (Lanza et al., 1987). Nutrition recommendations for diabetic subjects suggest that the level of fibre intake should be about 40 g per day (Canadian Diabetes Association, 1989; American Diabetes Association, 1992). Daily consumption of a diet containing both insoluble and soluble fibre from a wide variety of food sources is recommended; soluble fibre is thought to have a beneficial effect on both blood glucose and lipid levels.

The present study was designed to evaluate the long term effect of a dietary fibre intake of 40 g per day on metabolic control of NIDDM subjects as measured by dietary, clinical and biochemical methods. To achieve a high intake of dietary fibre, yeast bread/bread products containing oat bran concentrate (high in soluble fibre) were incorporated in the usual dietary pattern of each subject. Each period of the study, the OBC period and the WB period, was 12 weeks in length. The study was a randomized crossover design which allowed the subjects to serve as their own controls. A possible disadvantage of the crossover design is a carry-over or period effect (Woods et al., 1989). However, statistical analyses of the data from the present study indicated that the sequence of the diets had no significant effect on the results; no group by period interaction effect was evident.

The oat bran concentrate product used in the present study was obtained from Finland; it had a dietary fibre content of 45% and a ß-glucan content of 22.8% (Malkki, 1992a). In the yeast bread formula developed, oat bran concentrate was utilized at a level of 45% to replace wheat flour. Undesirable quality attributes resulting from the incorporation of oat bran concentrate into the bread included: a weakening of the gluten structure, poor volume, development of off-flavor and an unpleasant mouthfeel. The quality of the bread was optimized by modifying procedures and formulation; ingredients added included vital wheat gluten, fat and a surfactant (sodium stearoyl-2-lactylate). Formulations for buns and muffins of acceptable quality were also developed.

The oat bread concentrate bread products were well accepted by the subjects. The objective of the study was to incorporate eight slices of bread, containing oat bran concentrate, into the diets of the subjects to obtain a dietary fibre intake of over 30 g per day. In the OBC period, the actual intake of the subjects was 5.7 slices of oat bran concentrate bread per day, 71% of the target. The mean dietary fibre intake was 34 g per day and the oat bran concentrate supplied 8.6 g soluble fibre per day.

Subject compliance with the study protocol was very good. The researcher provided the intensive diet counselling required to implement and sustain the prescribed diet. Using a yeast bread as a vehicle for fibre helped to obtain dietary

compliance. A bread product with significant nutritional value as well as desirable quality minimizes changes in traditional food selection; in addition, the buns and muffins containing oat bran concentrate added variety to the diet. Dietary adherence was also promoted by individualizing the diet plans and meeting regularly with each subject, and spouse if appropriate (Kushner, 1993).

Body weight remained unchanged during the study periods. The average change in body weight for the whole group was 1.5 ± 0.4 kg for the WB period and 1.2 ± 0.4 kg for the OBC period. These changes were non significant. There were no differences between periods in the intake of carbohydrate or fat; mean dietary fat intake was constant at 30% of kcal. Saturated fatty acid intake remained unchanged; 10% of kcal for the WB period, 9% of kcal for the OBC period. The P/S ratios for the two periods were similar, 0.7 and 0.8. Changes in body weight and fat intake have been shown to affect carbohydrate and lipid parameters (Anderson et al., 1984a; Coulston, 1994).

In this study, the level of mean energy intake was comparable to that reported for males, 40 to 64 yrs of age, in the Nutrition Canada (Health and Welfare Canada, 1973) survey (2671 kcal). For the OBC and WB periods mean energy intake was 2658 and 2411 kcal, respectively. In each period the researcher assessed dietary intake quantitatively for a total of eight days to obtain a representative estimate of usual

food intake. Young et al. (1953) compared the seven-day intake of a group of adults to a 28-day intake and found that the seven-day intake was representative of the 28-day period. The dietary assessment at study entry provided background information about the subject's pattern of eating and enabled us to verify details and avoid incomplete data collection. The use of repeated recalls, using standardized interview techniques, enabled us to obtain reliable estimates of usual intake without requiring the subject to keep records which can distort a subject's customary food pattern. Errors in evaluating foods consumed were minimized because the researcher is a dietitian, knowledgable about foods, food composition, food preparation, meal patterns, food habits and local customs. Precision in data collection was facilitated by the use of standardized three-dimensional models to estimate serving portions and the use of Nutrition Canada methodology (Health and Welfare Canada, 1973).

At study entry, dietary intakes were assessed by only one 48-hour recall for the purpose of evaluating dietary patterns. This information was used for formulating individualized diet plans for each subject. Mean energy intake obtained was only 2000 kcal per day; a low energy intake was anticipated because quantitative assessment of energy intake requires more than two days of data. Madden et al. (1976) showed that under reporting is a common problem of the 24-hour recall and that false negatives were possible for values of energy, protein or

vitamin A intake. However, in the present research, entry data were very useful throughout the study for cross checking dietary information provided by each subject. Skilled probing for items not reported is a very important part of dietary assessment methodology.

The use of repeated recalls for assessing dietary intake throughout the study was appropriate methodology for relating food consumption to clinical status. Hawthorne (1981) showed that the increased precision required for correlating dietary intake to clinical status could be obtained by collecting quantitative dietary data from an individual over time. Researchers (Food and Nutrition Board, 1986) have reported that by increasing the number of days of collection from one to four, the confidence limits are reduced by one-half and the reliability of the estimate of usual dietary intake is improved. Keys (1979) and Yudkin (1951) felt that seven days of dietary data was the minimum to measure subject's customary consumption. Woteki et al. (1986) have used three-day intakes to examine various health and disease characteristics of the population. Dietary data in the present study were obtained for eight days in each period. Standardized Nutrition Canada methodology (Health and Welfare Canada, 1973) helped to minimize respondent or interviewer bias and improve the accuracy of food intake data (Young, 1981). We obtained multiple recalls on various days of the week to account for daily variation in food choices.

GLYCEMIC RESPONSE AFTER MEALS

Eight-hour plasma glucose day profiles were used in the current study to assess glucose metabolism. The eight-hour day profile provides a better assessment of the glycemic response of the subjects than the standard three-hour oral glucose tolerance test (National Diabetes Data Group, 1979). Gannon and Nuttall (1987) reported that, for NIDDM subjects, postprandial plasma glucose levels returned to fasting levels in four to five hours compared to 1.5 hours for normal subjects. The plasma glucose area under the curve (AUC) was calculated as the incremental response for eight hours (from 8 am to 4 pm), the integrated area above the baseline levels. The incremental area under the curve was calculated rather than the total area under the curve because the incremental AUC more accurately describes the glycemic response to food (LeFloch et al., 1990).

In the present study, the most striking finding was an improvement in glycemic response after meals in the OBC period compared to the WB period. OBC period total glucose incremental area under the curve was 41% lower ($p \leq 0.05$) than in the WB period. In the OBC period, glucose AUC after breakfast was 35% ($p \leq 0.01$) and after lunch was 50% lower (NS) than in the WB period.

The one compound that has proven to be effective in lowering postprandial plasma glucose levels of the diabetic subject is the soluble fibre, guar gum (Jenkins et al., 1976).

Jenkins et al. (1978a, 1980c) tested the guar gum alone compared to a glucose load and also guar gum in cereal products compared to wholemeal bread and cheese. The guar gum (14.5 g per day) alone flattened the glucose response so that the area under the curve was 50% lower than that for the glucose alone (Jenkins et al., 1978a). The guar gum in cereal products was tested with six diabetic subjects and in this case the area under the curve was 50% lower than for the control meal (Jenkins et al., 1980c). Ray et al. (1983) also reported lower postprandial glucose values in NIDDM individuals following guar ingestion. Ray et al. (1983) showed that 20 g guar at breakfast reduced the four-hour glucose profile compared to 10 g wheat bran at breakfast. Najemink et al. (1984) found the ingestion of 15 g guar in minitablet form significantly lowered the one-hour postprandial glucose response for NIDDM subjects after breakfast.

The physiological response obtained from guar gum was attributed to the viscosity of the product. High viscosity is thought to act as a barrier preventing the contact of digestive enzymes with their substrates and thickening the unstirred water layer near the mucosa causing a delay in glucose absorption. Jenkins coined the term "lente carbohydrate" to describe this effect (Jenkins et al., 1982). However, measurement of viscosity in vitro does not indicate viscosity within the gastrointestinal contents. Jenkins et al. (1978a) found that the improvement in glycemic response

correlated with the viscosity of the guar gum and that hydrolyzed non-viscous guar had no effect. However, Edwards et al. (1987) found no significant differences in impact on glycemic response between gums of widely different viscosities. Tinker and Schneeman (1989) have shown that viscous contents in the intestinal lumen do, in fact, limit the rate of digestion by decreasing access of digestive enzymes. It is apparent that products with a high viscosity can delay the absorption of starch from the small intestine. This, however, is not a property of soluble fibres in general.

ß-glucan gum, the main soluble fibre in oats, has a high viscosity and has potential for improving glycemic control. Braaten et al. (1991) found that oat gum significantly reduced plasma glucose excursions compared to a glucose drink; the effect of oat gum and guar gum were found to be similar. However, for every day diets, oats provide a more palatable source of soluble fibre than guar gum. Rating of the oat bran concentrate breads by subjects in the current study showed they were well accepted and 75% of the subjects were willing to buy the products if commercially available. Subjects reported that side effects were minimal. In the current study OBC period glucose excursions were reduced after meals; by 14% after breakfast (peak #1) and 25% after lunch (peak #2) compared to the WB period. In addition, OBC period glucose peak values were lower than in the WB period; 13% for peak #1 and 15% for peak #2.

Although the high viscosity of oat gum is thought to be responsible for its measured effect, the mechanisms responsible for the beneficial effect of oat gum on glycemic response require further investigation. Illman and Topping (1985) found that glucans from different cereals have different physiological effects. Not much is known about the physiological effects of many components of dietary fibre. Various aspects now being investigated include the effects on digestion and absorption of carbohydrate and fat, intestinal production of short chain fatty acids, binding of bile acids, and effects on gut hormones. Components in oats, other than dietary fibre, may also have an impact on glucose metabolism. In addition, the way in which the oat product is prepared may play a role in the mechanism of action (Slavin, 1987). The particle size of a product, for example, has been shown to affect the physiological response. Jenkins et al. (1988b) showed that bread containing intact whole grains could reduce glycemic response but that bread containing flour milled from the wholegrains had little effect. Jenkins et al. (1983b) postulated that naturally occurring fibre may hinder the penetration of food by digestive enzymes reducing the rate of digestion, and, that milling of wheat may disrupt the relationship of starch and fibre, thus reducing any effect of fibre on postprandial glycemia. However, Heaton et al. (1988) found that this principle did not apply to oat products. Heaton et al. (1988) showed that particle size had little

effect on metabolic response to oat based meals but had a significant effect on wheat and maize based meals. In contrast, Snow and O'Dea (1981) observed that, for many grains including oats, particle size and surface area to starch ratio were important factors influencing the availability of starch to hydrolytic enzymes. In the finely ground form, cereals were hydrolyzed more quickly. The oat bran concentrate used in the present study was a fine powder, much like flour. Snow and O'Dea (1981) noted that mechanical barriers such as a protein matrix (that encapsulates gelatinized starch molecules and limits access to enzymes) may contribute to a reduced glycemic index. Bread consists of starch embedded in a gluten matrix with fat lubricating the gluten. Bread is lower in gluten than spaghetti and has a higher glycemic index than spaghetti. The oat bran concentrate bread formulation in the present study contained added vital wheat gluten as well as other ingredients (e.g. surfactants such as sodium stearoyl-2-lactylate) to ensure an acceptable product. Thus, ingredients in the oat bran concentrate bread formulation may have acted as a physical barrier and decreased exposure of the starch to digestive enzymes. The oat bran concentrate was ingested primarily as bread (mean intake per subject was 5.7 ± 0.6 slices per day for the OBC period compared to 5.9 ± 0.6 slices per day for the WB period). Snow and O'Dea (1981) reported that starch in bread hydrolyzed faster than starch in cooked cereal. These researchers also noted differences in the rates

of hydrolysis of starch from homemade and commercial breads. In the present study there may also have been some hydrolysis of ß-glucan in oat bran concentrate by ß-glucanases in the malt flour in the bread recipe (Malkki, 1992a).

The literature reports conflicting results, even with guar gum which has proven to have a beneficial effect on glycemic response. Beattie et al (1988) conducted a long term crossover study (20 wk) with NIDDM subjects to test the effect of guar gum supplementation (15 g per day) compared to a placebo. The guar gum had no effect on the control of blood glucose and had an undesirable effect on the gastrointestinal tract resulting in increased diarrhea and flatulence. The study was well controlled; body weight did not change throughout the study. Similarly, Uusitupa et al. (1989) reported that the use of guar gum (15 g per day taken three times per day with meals) in the long term treatment of NIDDM subjects had no beneficial effect.

At present there is confusion about the most appropriate method of analyzing dietary fibre. Initially fibre content of food was determined as crude fibre. However, crude fibre does not represent most of the indigestible material in foods as was once thought and more recently analyses of dietary fibre have replaced analyses of crude fibre. At present there is still controversy regarding the most appropriate method for analysis of dietary fibre (Li and Andrews, 1988). Although different analytical methods (gravimetric, chromatographic,

colorimetric) give comparable values, differences in the methods used to separate soluble and insoluble components influence the results. In the present study, dietary fibre data were obtained from McCance and Widdowson's food composition table (Paul and Southgate, 1978); these data represent unavailable carbohydrate components remaining in the alcohol-insoluble residue after enzymatic hydrolysis of starch (Southgate, 1977). In the present study, the mean dietary fibre intake increased from 18.5 g/d in the WB control period to 33.5 g/d in the OBC period. The recently revised nutrition recommendations for people with diabetes (American Diabetes Association, 1994) suggest that the daily diet contain 20 to 35 g of dietary fibre.

The testing of glycemic response to naturally occurring fibres in foods has produced conflicting results. The issue of whether or not high fibre diets have a beneficial effect in the treatment of the NIDDM subject is controversial. Anderson's group (Kiehm et al., 1976) were among the first to report the benefits of a high fibre, high carbohydrate diet in the treatment of NIDDM subjects. Kiehm et al. (1976) demonstrated significantly reduced fasting blood glucose in 10 diabetic men consuming a diet containing 75% carbohydrate and 14.2 g crude fibre per day (the control diet contained 43% of kcal from carbohydrate, 4.7 g crude fibre). For the five NIDDM men in the study, treatment with sulfonylureas was discontinued after two weeks on the diet. Anderson and Ward

(1978) reported that a diet containing a large amount of fibre (36 g dietary fibre/1000 kcal) improved glycemic response and that, for some of the subjects, use of hypoglycemics decreased. The distribution of kilocalories in the diet was 70% carbohydrate, 19% protein and 11% fat. The diet produced lower values for both postprandial and fasting plasma glucose. However, the subjects lost weight, a factor known to improve glycemic response. The high fibre diets which have had beneficial effects on glycemic control of diabetic subjects have contained high amounts of fibre and low glycemic index foods such as legumes (Kiehm et al., 1976; Jenkins et al., 1980c; Simpson et al., 1981; Jenkins et al., 1983a; Riccardi et al., 1984). In general, the successful dietary regimes contain more soluble dietary fibre components than the unsuccessful dietary regimes (more high insoluble fibre cereal foods) (DelToma et al., 1988a, 1988b; Chew et al., 1988).

The glycemic response to a particular food correlates only weakly with dietary fibre content. Other factors are important; e.g. the chemical and mechanical forms in which carbohydrate is held, the characteristics of the starch and the presence of antinutrients (Indar-Brown et al., 1992). For example, Jenkins et al. (1983b) demonstrated in nine diabetic volunteers that food form, independent of fibre content, affects glycemic response. Blood glucose rise following ingestion of 50 g white or wholemeal bread was identical but glucose response after 50 g white spaghetti was markedly

reduced (Jenkins et al., 1983b). Compared with the mean 3-hour response to bread, the spaghetti blood glucose area response was reduced by 35% ($p \leq 0.001$) and the peak rise by 42% ($p \leq 0.001$). Jenkins et al. (1983b) suggested that the milling of wheat products may possibly disrupt the normal relationship of starch and fibre, thus reducing any effect of fibre on postprandial glycemia. In addition, protein/carbohydrate complexes can be formed in the production of spaghetti limiting starch availability (Bornet et al., 1987). Krezowski et al. (1987) reported that a 10% greater plasma glucose area under the curve might be anticipated for starch containing foods compared to an equivalent mass of glucose, because starch consists of anhydrous glucose units.

The glycemic response to meals is affected by many factors including the fat and protein in the meal (Collier and O'Dea, 1983; Nuttall et al., 1984, 1985; Collier et al., 1984), the rate of ingestion, the gastric emptying rate, and the release of regulatory gut hormones (Nuttall, 1993). A carry-over effect has also been noted after the ingestion of soluble fibre, with a blood glucose lowering effect being obtained both after the meal containing the soluble fibre and after the following meal (Jenkins et al., 1980a, 1982; Wolever et al., 1988).

In studies reporting improved carbohydrate metabolism with increased fibre, interpretation is usually complicated by other changes such as weight loss (Kiehm et al., 1976; Simpson

et al., 1981), decreased total food energy intake (Kiehm et al., 1976), and inclusion of large amounts of poorly digested legumes (Kiehm et al., 1976; Simpson et al., 1981; Nuttall, 1993). Many studies evaluated the effects of large intakes of fibre, e.g. 70 g dietary fibre per day (Anderson and Ward, 1978; Simpson et al., 1981). Some well controlled studies have used diets containing about 40 g dietary fibre per day based on the traditional food habits of the North American. Many of these studies have failed to show a beneficial effect on glycemic response in NIDDM subjects. Hollenbeck et al. (1986a) found that increasing fibre intake from 11.0 to 22.7 g/1000 kcal/d had no significant effect on fasting or postprandial plasma glucose or on insulin levels. Coulston et al. (1987) found that 38 g fibre per day in a high carbohydrate diet (60% of energy from carbohydrate) resulted in deleterious metabolic effects in patients with NIDDM. There was significant deterioration in glycemic response after only 15 days on this diet. Similarly, in 33 NIDDM outpatients, Scott et al. (1988) found a deterioration in blood glucose control with an increase in mean fibre intake of 16 g per day. The subjects were on a high fibre diet for 6 mo. Deterioration in diabetic control was greatest in those who ate most fibre. For some NIDDM patients insulin reserve has declined and as a result glucose tolerance is poor and a high carbohydrate intake may raise plasma glucose levels (Grundy, 1991).

In the present study there was no improvement in fasting plasma glucose levels. Coulston et al. (1987) found that with an increase in the level of carbohydrate in the diet from 40 to 60% of kcal, fasting plasma glucose values increased. The recent nutrition recommendations and principles for people with diabetes mellitus (American Diabetes Association, 1994) state that first priority should be given to the total amount of carbohydrate consumed. In addition, fasting plasma glucose levels in NIDDM individuals are very sensitive to alteration in total caloric intake (Savage et al., 1979). Weight reduction will improve glycemic response (Wolever et al., 1992; Wing et al., 1994). The design of the present study specified weight maintenance, even if the subject was overweight, so that the effects of oat bran concentrate could be evaluated. Weight reduction for the overweight NIDDM subject is crucial for good control of diabetes. In the current study, glycosylated hemoglobin was monitored to follow the level of plasma glucose control. This form of hemoglobin reflects the average blood glucose concentration over a three month period. In the present study there was no improvement in glycosylated hemoglobin levels. Singer et al. (1989), in an evaluation of glucose parameters, concluded that glycosylated hemoglobin levels may take longer to show an effect than either plasma glucose or insulin levels.

The present study showed that a long term diet containing 34 g dietary fibre per day improved diabetic control. The

level of carbohydrate in the diet was 50% of kcal. Incorporating 9 g of soluble fibre in the form of oat bran concentrate in the usual dietary pattern of NIDDM individuals (mean total dietary fibre intake of 34 g) improved glycemic response significantly compared to the control diet with a dietary fibre intake of 19 g per day. In the OBC period, glucose area under the curve was 41% ($p \leq 0.05$) lower than in the WB period. OBC period glucose peak values were lower than in the WB period; 13% lower after breakfast and 15% lower after lunch. There was less hyperglycemia overall. The bread group's contribution to total fibre intake increased from 43% in the WB period to 70% in the OBC period. In comparison, for the average North American male (Block and Lanza, 1987) dietary fibre intake is ~13 g per day and the bread group supplies 24% of the fibre. The high carbohydrate, high fibre maintenance diet used so successfully by the Anderson group (Kiehm et al., 1976) contains over 45 g dietary fibre per day; 40% of this fibre is derived from the bread group.

In the present study, the achievement of better glycemic control with the incorporation of oat bran concentrate in the diet is of great benefit to the NIDDM subject. The recent Diabetes Control and Complications Trial (DCCT) (American Diabetes Association, 1993) has demonstrated the value of maintaining as good glycemic control as possible. Hyperglycemia is associated with glycosylation of protein molecules, and microvascular complications including

retinopathy, nephropathy and neuropathy. The DCCT study has shown a clear relationship between improved glycemic control and a lessening of complications for the insulin dependent diabetic patient. For the NIDDM diabetic individual lowered glucose levels should also decrease the incidence and progression of complications. Recent evidence indicates that chronic hyperglycemia leads to progressive impairment in insulin secretion, impairment of insulin mediated glucose transport and may contribute to insulin resistance (Rossetti et al., 1990). The improvement of glycemic control by dietary means could greatly benefit the overall health of the non-insulin-dependent diabetic individual.

INSULIN RESPONSE

In the present study, the postprandial insulin response was less in the OBC period than in the WB period. The total area under the insulin curve after the meal challenges (eight-hour day profile) was 17% (NS) lower in the OBC period than in the WB period. The area under the insulin curve after the breakfast meal (four-hour day profile - part #1 of the eight-hour day profile) was significantly lowered 22% ($p \leq 0.05$); the area under the insulin curve after the lunch meal (four-hour day profile - part #2) was 13% lower than in the WB period. The improvement in insulin secretion was associated with an improvement in glucose metabolism as noted by the improved glycemic response in the OBC period compared to the WB period.

The OBC period peak insulin values were lower than in the WB period; 15% lower for peak #1 and 11% lower for peak #2.

Hyperglycemia after eating is the major stimulus to insulin secretion. In subjects with NIDDM, insulin secretory failure and impaired insulin action are both present; the underlying primary metabolic abnormality is poorly defined. Insulin resistance in the NIDDM subject leads to compensatory hyperinsulinism. After a glucose challenge, insulin values return to baseline more slowly in NIDDM individuals compared to non-diabetic persons. Gannon and Nuttall (1987) observed that, following ingestion of 50 g glucose, in non-diabetic volunteers, insulin concentration returned to baseline values in three hours, while in the NIDDM individual insulin concentration was still slightly elevated after five hours. Thus, in the present study, the eight-hour day profiles allow adequate time to evaluate insulin response.

For the NIDDM subject, insulin response is not sufficient in proportion to the level of glycemia. However, non-insulin-dependent diabetes is a heterogenous syndrome; during day profiles insulin concentrations vary widely, increasing with increasing peripheral resistance. The response of pancreatic ß-cells is a very individual characteristic and it is possible to induce an increase in pancreatic sensitivity. Weight loss, for example, leads to improved insulin sensitivity of ß-cells to insulinogenic stimuli, and enhanced glucose tolerance. Three of the eight subjects in the present study were taking

oral hypoglycemic agents (sulfonylureas) to stimulate the release of preformed insulin from the pancreas. In the present study there was evidence of increased sensitivity of ß-cells in the OBC period as compared to the WB period. OBC maximum insulin values were reached earlier than for the WB period, 26 minutes ($p \leq 0.05$) earlier for peak #1 and 30 minutes earlier for peak #2. In non-diabetic persons, peak insulin values are usually reached one-half and one hour after a glucose challenge while in diabetic individuals it may be nearer two hours. In the present study the earlier insulin peak with oat bran concentrate suggests that perhaps ß-cell function improved. Cohen et al. (1990) reported that a meal with a low glycemic response resulted in an earlier insulin peak than a meal with a high glycemic response. These researchers evaluated the insulin and glycemic responses to a high-glycemic-effect meal (including cornflakes, bread) and a low-glycemic-effect meal (including oatmeal, fructose). The subjects were six obese non-insulin-dependent diabetic individuals. The integrated insulin response areas (5-hour profiles) of the two meals did not differ but the peak insulin level occurred 60 minutes earlier in the low-glycemic-effect meal.

In the present study, postprandial insulin response (total incremental area under the curve) was negatively correlated ($p \leq 0.05$) with fibre intake, both total dietary fibre and soluble fibre. One explanation for the lower insulin

response in the OBC period than in the WB period is that the rate of glucose absorption is slowed in the presence of the viscous oat bran concentrate soluble fibre and a smaller amount of insulin is released. Resistant starch, with properties similar to those of dietary fibre, may play a role in the mechanism of action. Jenkins et al. (1978a) showed that ingestion of the soluble fibre guar resulted in an insulin response that was 50% lower than that obtained with glucose. Kiehm et al. (1976) were among the first to report that a high fibre, high carbohydrate diet had a beneficial effect on NIDDM subjects; for all five NIDDM subjects who were on oral hypoglycemics (sulfonylureas), treatment with sulfonylureas was discontinued when the high fibre, high carbohydrate diet was consumed. Recent work (Fukagawa et al., 1990) suggests that, in addition to the impact of the intestinal phase of glucose uptake, there is also a change in the responsiveness of peripheral tissues to insulin-mediated glucose uptake. In healthy young and old individuals, these researchers showed that the high-carbohydrate (68% of kcal from carbohydrate), high-fibre (35 g dietary fibre/1000 kcal) HCF diet for three to four weeks increased significantly the sensitivity of peripheral tissues to insulin compared to the usual diet. To examine the effects of the HCF diet they measured insulin-mediated glucose disposal employing the euglycemic clamp and measurement of hepatic glucose output. The study showed no change in basal hepatic glucose output during the HCF diet

period but rather an increase in the sensitivity of peripheral tissues to insulin. One study (Pedersen et al., 1990) reported that fibre may modify the binding of insulin to receptors.

In the literature there is a controversy regarding the impact of soluble fibre on insulin response. DelToma et al. (1988a, 1988b) noted that soluble fibre lowers insulin response. Hollenbeck et al. (1986a), however, reported that insulin response did not change with a change in total dietary fibre intake of 11 g to 27 g/1000 kcal. In studies using the soluble fibre guar, Jenkins et al. (1982) suggested that postprandial insulin response can be predicted by post-meal glucose response. Jenkins et al. (1978a, 1980c) demonstrated that guar supplementation, compared to a glucose load, flattened both glucose and insulin response by 50%. However, insulin response to meals does not always parallel the glucose response as predicted. Krezowski et al. (1987) observed that oatmeal in particular does not act as expected and "oatmeal ingestion resulted in the greatest mean insulin area when compared with other high-starch foods". The subjects for his study were eight non-insulin-dependent diabetic persons. The glucose response area for 50 g carbohydrate as oatmeal was similar to that for bread and rice and lower than that for glucose, but insulin area for oatmeal was 125% greater than for glucose. Oatmeal ingestion produced the greatest mean absolute insulin area when compared with other high starch foods (Krezowski et al., 1987). The reason for the increased

insulin response to oatmeal is not known; it was postulated that differences in the digestibility of the starch molecule of oats may be a factor in the increased insulin response.

Insulin response to meals is influenced by many factors. Chew et al. (1988) attributed some of the variation to the impact of other nutrients in the meal such as protein which acts as an insulin secretagogue. Indar-Brown et al. (1992) reported that, for NIDDM subjects, the insulin response to meals differed and Nuttall and Gannon (1991) suggested that, for the NIDDM subject, protein had a greater impact on insulin secretion than for the non-diabetic subject. The level of fat intake affects insulin response. High fat diets have an impact on insulin response by causing insulin resistance and impairing intracellular glucose metabolism (Anderson, 1988). Nuttall and Gannon (1991) found insulin response area ~45% greater when fat (butter) was ingested with potato compared to potato alone. Dietary fat decreases the number of insulin receptors in several tissues and decreases activities of insulin stimulated processes (Anderson, 1988). Randle et al. (1963) hypothesized that an inverse relationship exists between circulating free fatty acid levels and insulin-mediated glucose disposal.

Many other factors affect insulin response including the nature of the carbohydrate (amylose, amylopectin) (Behall et al., 1989), the presence of starch-nutrient interactions, food processing. The oat bran concentrate bread in the present

research contained additional vital wheat gluten and other ingredients which may have insulated embedded starch and ß-glucan from enzyme hydrolysis.

Morgan et al. (1979) suggested that the gut hormone, gastric inhibitory polypeptide (GIP), may be partly responsible for the smaller rise in plasma insulin observed in normal subjects when guar is added to meals. Morgan et al. (1979) measured insulin/glucose ratios by calculating the ratio of the peak plasma insulin value to the corresponding blood value. The researchers found that the insulin/glucose (I/G) ratio decreased from 6 to 4 when guar was added to meals and that GIP played a significant role in the reduction of postprandial insulin secretion by guar. They hypothesized that the rate of glucose absorption regulated the release of GIP from the gut which in turn stimulated insulin release. The effect of guar on the rise of GIP levels was found to be greater when added to a carbohydrate meal than to a mixed meal (Morgan et al., 1979). A decrease in the plasma I/G ratio was observed with oat gum compared to glucose alone (Braaten et al., 1991). In the present study, insulin/glucose ratios were similar for the OBC and WB period. For peak #1 (breakfast), the insulin excursion was lower (NS) in the OBC period than in the WB period. The results of the day profiles showed that the areas under the curves (AUC) following breakfast and lunch were decreased more for plasma glucose than for serum insulin. For the four-hour breakfast profile, the OBC period insulin

AUC was 21% lower and the glucose AUC was 35% lower than in the WB period. For the four-hour lunch profile, the OBC period insulin AUC was 13% lower and the glucose AUC was 50% lower than in the WB period. The greater reduction in glucose than insulin response may indicate enhanced ß-cell sensitivity to insulinogenic stimuli.

Recently, evidence is accumulating to indicate that the excess glucose present in the plasma of the NIDDM subject can have adverse effects on the pancreas. Chronic hyperglycemia can result in decreased pancreatic function and changes in pancreatic structure (Rossetti et al., 1990). The mechanism by which chronic hyperglycemia causes ß-cell blindness to the stimulatory effect of glucose on insulin secretion is being explored extensively. There is evidence that chronic hyperglycemia may contribute to insulin resistance as well. In the present study the improved glycemic response of the subjects may have contributed to enhanced ß-cell function. In NIDDM patients, preservation of the ß-cell function is of importance. Yki-Jarvinen (1992) concluded that insulin sensitivity improves with glycemic control and, for the NIDDM diabetic individual, insulin secretion is amenable to improvement.

In the present study there was no change in either the mean fasting insulin value or the mean fasting glucose level. In this study diets were controlled to maintain body weight to allow evaluation of the effect of oat bran concentrate on

metabolic parameters. Body weight did not change in this study even though many subjects were overweight; relative body weights ranged from 100% to 157%. For the NIDDM subject an important principle of treatment is the achievement and maintenance of a reasonable body weight. For the overweight subjects, loss of weight will lead to a fall in fasting glucose concentration by depleting liver glycogen stores, and decreasing hepatic glucose output (Henry et al., 1985). Yki-Jarvinen and Taskinen (1988) have shown that hypertriglyceridemia is often associated with non-insulin-dependent diabetes and that the regulation of lipoprotein lipase is abnormal in the insulin resistant, obese, hypertriglyceridemic subject. NIDDM subjects with hypertriglyceridemia are resistant to both the glucoregulatory and the antilipolytic actions of insulin.

In the present study, even though body weight did not change, insulin response of the NIDDM subjects was improved in the OBC period compared to the WB period. Further research is required to clarify the role of dietary fibres and other components in oat bran concentrate on glucose/insulin interrelationships in diabetic individuals.

LIPID RESPONSE

In the present study, oat bran concentrate (8.6 g soluble fibre per day) reduced plasma cholesterol levels by 14% ($p \leq 0.01$). The plasma total-C was 5.30 ± 0.11 mmol/L for the WB

period, 4.56 ± 0.11 mmol/L for the OBC period. The cholesterol content of the LDL fraction decreased from 3.36 ± 0.12 mmol/L in the WB period to 2.59 ± 0.12 mmol/L in the OBC period, representing a decrease of 23% ($p \leq 0.01$). Similar hypolipidemic effects have been obtained with a variety of soluble fibres such as guar gum, oat bran, oatmeal and legumes (Anderson et al., 1984a, 1984b, 1990a, 1990b; VanHorn et al., 1986, 1988, 1991; Lepre and Crane, 1992; Uusitupa et al., 1992). Shinnick et al. (1990) demonstrated a dose response relationship between oat fibre intake and lipid lowering. In an animal study, ingestion of 4% and 10% dietary fibre from oat bran decreased serum cholesterol 15% and 28%, respectively ($p \leq 0.05$). Ripsin et al. (1992) evaluated the lipid lowering effect of oats (but not oat bran concentrate) and concluded that 3 g soluble fibre can lower total-C from 0.13 to 0.16 mmol/L. For the amount of oat bran concentrate used in this study (8.6 g soluble fibre per day) the predicted decrease in total-C would have been from 0.37 to 0.45 mmol/L, much less than the decrease achieved, 0.74 mmol/L. It has been suggested that the soluble fibre in oat bran concentrate may be unique and more effective than oat bran (Ranhotra et al., 1990).

The initial level of plasma cholesterol was found to be predictive of the lipid lowering effect of soluble fibre (Ripsin et al., 1992). Following oat bran consumption, Anderson et al. (1991) achieved a cholesterol reduction of 12.8% in subjects with an initial cholesterol level of 6.9

mmol/L. VanHorn et al. (1991) found a greater reduction in total-C in individuals with a baseline median serum total-C of 6.34 mmol/L than in individuals with baseline values below 6.34 mmol/L. In the present study, the mean plasma cholesterol level at study entry was in the normal range. However, Stamler et al. (1986) reported that the risk of cardiovascular disease is linearly related to serum cholesterol level even when the values are within the normal range. Because the risk of coronary heart disease is increased in diabetic subjects, a reduction of serum cholesterol, particularly its LDL fraction, should contribute to a long-term positive prognosis in these individuals (Kannel and McGee, 1979). Serum cholesterol lowering with oat bran consumption has been demonstrated in both hyper- and normo-cholesterolemic subjects (Gold and Davidson, 1988). In the present study, the range of plasma cholesterol values at study entry was from 3.10 to 7.31 mmol/L. Oat bran concentrate reduced the total-C in each of the subjects regardless of whether initial level was normal or high. In contrast, Swain et al. (1990) found no cholesterol lowering in subjects with an initial cholesterol of 4.8 mmol/L and less.

In NIDDM subjects frequently total-C and LDL-C are not elevated (DeFronzo and Ferrannini, 1991; Hollenbeck and Coulston, 1991). Dyslipidemia of diabetes most commonly consists of decreased HDL-C and elevated TG levels (DeFronzo and Ferrannini, 1991; Coulston, 1994). HDL-C is a

cardioprotective lipid fraction (Gordon et al., 1977; Kirby et al., 1981; Anderson et al., 1984a, 1984b); a low HDL-C level has been reported to be a better predictor of cardiovascular disease risk than increased total-C (Denmark-Wahnefried et al., 1990). Subjects in the present study had low mean HDL-C levels (25^{th} percentile). The mean HDL-C levels of subjects did not change significantly; there was a slight (+8%) increase in HDL-C levels in the OBC period, indicating an HDL-C sparing effect. In other studies, soluble fibre (oat bran) ingestion has been shown to spare or actually raise HDL-C levels (Kirby et al., 1981; Rivellese et al., 1983; Anderson et al., 1984a, 1984b; Riccardi et al., 1984; O'Dea et al., 1989). HDL-C is not as sensitive to change as total cholesterol; Jenkins et al., (1988c) reported an increase in HDL-C in 24 weeks.

Hypertriglyceridemia is common in NIDDM subjects (DeFronzo and Ferrannini, 1991) and is associated with an increased risk for cardiovascular disease (Coulston, 1994). The WHO Multinational Study (West et al., 1983) and the Paris Prospective Study (Fontbonne et al., 1989) found that an elevated triglyceride level was the single risk factor most strongly correlated with cardiovascular disease in diabetic individuals (Hollenbeck and Coulston, 1991). In the present study there was no significant change in plasma triglyceride levels. However, there was a trend towards a decrease in triglyceride levels; the triglyceride levels in the OBC period were 5% lower than in the WB period and 10% lower than at the

beginning of the study. Several researchers have reported no change in triglyceride levels with oat products (Rivellese et al., 1983; Riccardi et al., 1984; O'Dea et al., 1989). Anderson and Bryant (1986) reported that high fibre high carbohydrate diets lower triglycerides and do not jeopardize triglyceride metabolism of most individuals with diabetes. A high carbohydrate diet may lead to increased serum triglyceride concentration because of increased hepatic synthesis of triglycerides from carbohydrate (Ginsberg et al., 1976). Energy balance is an important determinant of plasma triglyceride; elevated plasma triglycerides decrease with weight reduction (Anderson and Ward, 1978). We did not expect a lowering of triglycerides in our study because diets were designed to be weight maintaining and subjects' body weight did not change appreciably.

Several mechanisms have been proposed for the lipid lowering effect of oat products (Schneeman and Tietyen, 1994). Enhanced fecal bile acid excretion is one proposed mechanism. Anderson et al. (1984b) demonstrated that both oat bran and bean supplements fed to 20 hypercholesterolemic men for 21 days reduced serum total-C. Fecal bile acid excretion was increased with oat bran supplementation but did not change with bean supplementation (Anderson et al. 1984b). Therefore, oat bran enhances bile acid excretion but whether or not the mechanism involves soluble fibre is not known. The heterogeneity of the dietary fibre composition of foods

precludes any general conclusion. Kelley and Story (1987) demonstrated in animal experiments (rats) that oat bran had a direct effect on cholesterol metabolism and decreased the activity of HMG-CoA reductase. Oat bran, by delaying glucose absorption, decreases the release of insulin following a meal. Insulin has been found to directly stimulate sterol synthesis through the HMG-CoA reductase pathway (Keenan et al., 1991). Therefore, cholesterol synthesis in the liver may be reduced by lowered postprandial glycemia and insulinemia (Brand Miller, 1994). Serum insulin levels may affect both cholesterol and triglyceride levels (Anderson et al., 1987). Insulin regulates both cholesterol and triglyceride synthesis; fasting triglycerides improve with enhanced glucose control (Brand Miller, 1994).

Another possible mechanism is the effect of short chain fatty acids (acetate, propionate and butyrate) produced by soluble fibre fermentation in the large bowel. Short chain fatty acids are absorbed through the portal vein and can inhibit hepatic cholesterol synthesis (Anderson et al., 1984b; Anderson and Gustafson, 1988; Schneeman and Tietyen, 1994). In addition, propionate may stimulate glycolysis and facilitate glucose use (Anderson et al., 1987). In the human, oat bran was shown to significantly increase serum acetate levels over a 14 hour period (Bridges et al., 1992). Slowing the rate of lipid absorption may have a beneficial effect on plasma lipids. The interaction of soluble fibre with bile acids and

phospholipids may slow lipid digestion and absorption because bile acids and phospholipids will not be available for micelle formation in the small intestine (Schneeman and Tieyten, 1994). Oat gum has been shown to reduce the uptake of cholesterol in the rat (Lund et al., 1989).

There are many confounding factors affecting the results obtained in studies of lipid lowering including: concommitant changes in fat intake, in body weight, and whether or not there was a control group. Several researchers have suggested that a reduced fat intake or a substitution of carbohydrate for dietary fat contributes to the lipid lowering (VanHorn et al., 1986; Jenkins, 1988; Denmark-Wahnefried et al., 1990; Swain et al., 1990). In their study, VanHorn et al. (1986) examined the cholesterol lowering properties of rolled oats (60 g/day) and concluded that rolled oats reduced serum cholesterol by 3% but that there was an additional 5% reduction from the use of a low fat diet. In the current study, dietary fat intake was similar in both dietary periods and did not affect lipid lowering.

Other dietary variables influence lipid lowering, including whether the total diet is self selected or clinically controlled. In the present study, oat bran concentrate (8.6 g soluble fibre per day) reduced plasma total-C levels by 14% and LDL-C levels by 23%, a significant hypolipidemic effect. A variety of results have been achieved with free-living subjects ranging from no change in blood

lipid levels (Leadbetter et al., 1991) to decreases in total-C of 2% to 12% (Storch et al., 1984; Keenan et al., 1991). In a metabolic ward setting, good lipid lowering has been achieved with diets high in soluble fibre; Kirby et al. (1981) found a 13% decrease in total-C, Anderson et al. (1984a, 1984b) demonstrated total-C decreases of 19% and 24%. Diabetic subjects with hyperlipidemia were reported to achieve good lipid lowering with a high fibre diet; for diabetic subjects, Rivellese et al. (1983) obtained LDL-C decreases of 30 to 35% compared to 10% for non-diabetic subjects.

In diabetic as in non-diabetic subjects, cardiovascular disease risk is directly proportional to LDL-C and inversely proportional to HDL-C. In the present study, lipid ratios were altered favorably. OBC period total-C/HDL-C and LDL/HDL ratios were lower by 14% (NS) and 25% ($p<0.05$), respectively, than in the WB period. In the Framingham Study (Castelli et al., 1983), the single best predictor of coronary heart disease was the ratio of total-C/HDL-C (Kestin et al., 1990). Total-C/HDL-C values of ≤ 4.5 are considered desirable (Denmark-Wahnefried et al., 1990). In the OBC period of the present study, total-C/HDL-C ratio was 4.4, less than the ratio of 5.5 in the WB period. Denmark-Wahnefried et al. (1990) found that the ratio of total-C/HDL-C decreased significantly over time in hypercholesterolemic subjects consuming OB supplemented diets. An LDL/HDL ratio of ≤ 3.5 is associated with reduced risk of atherosclerosis (Aro et al., 1981; Castelli et al., 1983;

O'Dea et al., 1989). In the OBC period of the present study, LDL-C/HDL-C ratio was 2.7, less than the ratio of 3.5 in the WB period. High fibre diets have been reported to improve the LDL/HDL ratio of NIDDM subjects (Aro et al., 1981; Castelli et al., 1983; O'Dea et al., 1989). Riccardi et al., (1984) found a lower LDL/HDL ratio in NIDDM subjects after a high carbohydrate (53%), high fibre (54 g per day) diet than following a low carbohydrate (42%), low fibre (20 g per day) diet.

Numerous studies have confirmed that the ingestion of high fibre diets or oat bran incorporated into the diet have favorably influenced blood lipid levels. Studies in which no hypolipidemic effects with high fibre diets were found have: used small amounts of ß-glucan (Bremer et al., 1991; Mackay and Ball, 1992), examined non-fasting blood cholesterol (Saudia et al., 1992), or had initial lipid values in the normal range (Swain et al., 1990). Some trials are confounded by significant weight loss for subjects (Jenkins et al., 1980b; Anderson et al., 1984a, 1984b; Denmark-Wahnefried et al., 1990); other studies did not include an adequate control group (Anderson et al., 1984a; Denmark-Wahnefried et al., 1990).

The present study shows that oat bran concentrate incorporated in the diet of NIDDM subjects can favorably influence plasma lipids. The incorporation of ~9 g soluble fibre per day in the diet resulted in significant lowering of

total cholesterol and LDL cholesterol levels. In addition, there was effective reduction of both the ratio of plasma total cholesterol to high-density lipoprotein cholesterol and the ratio of LDL-cholesterol to HDL-cholesterol. Diets containing oat bran concentrate have potential for decreasing the lipoprotein-mediated component of cardiovascular disease risk and improving the health of NIDDM individuals.

BREAD CONSUMPTION

In the current study, the oat bran concentrate as a source of soluble fibre in bread provided a mean intake of 8.6 g soluble fibre per day per subject. Subject intake of total dietary fibre increased dramatically (by 181%) as a result of the oat bran concentrate bread in the diet; the intake of total dietary fibre increased from 19 g in the WB period to 34 g in the OBC period. Oat bran concentrate bread product consumption improved both the glycemic response and blood lipid profiles of the NIDDM subjects.

Oat bran concentrate contains the gum ß-glucan (Wood et al., 1989a, 1989b). The viscous properties of oat bran concentrate present challenges in breadmaking. Problems of undesirable quality in yeast breads with oat bran concentrate (ie. reduced volume, gummy texture, unpleasant mouthfeel and off-flavor) were overcome by: using hard red spring wheat flour, the addition of vital wheat gluten (12%), a surfactant (0.5% sodium stearoyl-2-lactylate), shortening (3%) and

alterations in method (longer mixing and baking times). The substitution of forty-five percent oat bran concentrate for hard red spring wheat flour in the formulation resulted in a loaf with acceptable volume, small even cells, a moist but not gummy texture, a pleasant mouthfeel and a slightly nutty flavor. The oat bran concentrate bread was similar to whole grain bread in textural properties. Eating quality of the oat bran concentrate bread in the present study was excellent. The subjects ate the breads for three months.

In the present study, the oat bran concentrate, incorporated in bread/bread products, was taken as part of the subject's daily diet rather than as a supplement. No published studies have examined the incorporation of oat bran concentrate into yeast breads in the diet. Many studies examined the effect of oat bran or oatmeal incorporated into the diet via hot or cold cereal, biscuits, or muffins. Other researchers have put oat bran in a drink (Braaten et al, 1991) or distributed oat bran sachets with study-specific recipes (Mackay and Ball, 1992; Denmark-Wahnefried et al., 1990; Davidson et al., 1991; Leadbetter et al., 1991; Uusitupa et al., 1992). However, yeast bread is a staple in the North American diet; in Canada bread consumption is 65.18 kg per capita per year (Flax Council of Canada, 1994). Thus bread is more likely to be accepted by subjects long term.

In the present study reported side effects resulting from oat bran concentrate bread consumption (high soluble fibre)

were minimal. A feeling of fullness after eating the oat bran concentrate bread/bread products was the main comment and subjects noted some flatulence following oat bran concentrate bread consumption which diminished with time. Reported side effects with oat products appear to be minimal. Kirby et al. (1981) noted no gastrointestinal complaints from subjects when 100 g oat bran per day (14.8 g soluble fibre per day) for ten days was consumed. Some researchers have reported minor gas (belching and flatulence), cramping, bloating, and loose stools with oat fibre consumption (Swain et al., 1990; Stewart et al., 1992; Uusitupa et al., 1992; Keenan et al., 1991; Anderson et al., 1984b). Jenkins et al. (1978b) found flatulence to be minimal with guar crispbread (12 g per meal). In contrast, many NIDDM subjects ingesting the soluble fibre guar reported considerable flatulence, bloating and increased stool frequency (Aro et al., 1981; Ray et al., 1983; McIvor et al., 1985; Najemnik et al., 1984; Holman et al., 1987).

In the present research some of the ß-glucan in the yeast bread and buns used was partially hydrolyzed during bread making by the small amount of malt flour in the recipe (Malkki, 1992a); however, this did not appear to be detrimental to the effect of ß-glucan on the glucose response and lipid lowering. Viscosity of the oat bran concentrate bread in the present study was slightly higher than bread containing unconcentrated oat bran but lower than bread with uncooked oat bran concentrate added prior to the viscosity

analysis (Malkki, 1992a). Ranhotra et al. (1990) suggest that the soluble fibre in oat bran concentrate has a significant cholesterol lowering effect.

The desirable physiological effects from oat bran concentrate intake have been attributed to the ß-glucan content of the oat fibre (Chen et al., 1981; Jennings et al., 1988; Wood et al., 1989a, 1989b, 1990; Autio et al., 1992a, 1992b; Malkki, 1993). However, it is possible that other components (e.g. tocotrienols, arginine-lysine ratio) of oats may play a role (Davidson et al., 1991; Malkki, 1993). In addition, the preparation process of the oat product may affect the mechanism of action (Malkki, 1993). Particle size has been found to be a factor in glycemic response (Lewis, 1978; Jenkins et al., 1988b). However, Heaton et al. (1988) found that particle size did not make a difference with oats. The particles of oat bran concentrate in the present study were very small. Mechanical barriers such as protein matrix may contribute to reduced metabolic responses (Snow and O'Dea, 1981). Bread contains starch embedded in a gluten matrix with fat lubricating the gluten. In the current study, ingredients in the oat bran concentrate bread formulation may have provided a physical barrier and reduced exposure of the starch (ß-glucan) to digestive enzymes. The unique chemical structure

(non-starch polysaccharide ß-glucan plus resistant starch) and the physical properties of oat bran concentrate bread both may contribute to the physiological effects observed in this study.

SUMMARY AND CONCLUSIONS

This study evaluated the long term efficacy of incorporating oat bran concentrate bread products (eight bread exchanges = 25 - 30 g dietary fibre) in the usual dietary pattern of non-insulin-dependent diabetic (NIDDM) subjects via dietary, clinical and biochemical methods. Palatable high fibre oat bran concentrate yeast bread and buns and muffins (3 types) were developed and baked using oat bran concentrate (dietary fibre = 45%; ß-glucan = 22.8%) from Finland. Oat bran concentrate was incorporated into the bread products at a level of 45%. Deleterious effects on bread quality due to incorporation of a large amount of soluble fibre (ß-glucan) into yeast bread products were overcome by the use of vital wheat gluten, a surfactant and alterations in mixing and baking methods.

Eight NIDDM men, ($\bar{x}$ = 45 yrs), were selected according to specific criteria: diabetes controlled by either diet or a combination of diet plus oral hypoglycemic agents, BMI <35kg/m^2, glycosylated hemoglobin (HbA_{1C}) <10%, serum cholesterol (C) <7mmol/L and serum triglyceride (TG) <5mmol/L. No lipid lowering drugs were allowed. Subjects in the six-month study lived in the community. A crossover experimental design with two periods (12 weeks each) was used; each subject served as his own control. Four randomly chosen subjects ate oat bran concentrate bread first; the remainder ate control

white bread (WB) first. Individualized diet plans were formulated. Quantitative dietary data were obtained for eight days each for periods 1 and 2 and for two days at study entry. A Foods and Nutrition researcher used 48-hour dietary recalls to assess dietary intake; standardized interview techniques and 3-dimensional food models [Nutrition Canada methodology (Health and Welfare Canada, 1973)] were used. The researcher provided counselling to ensure long term dietary compliance. Patients were seen every three weeks for anthropometric and diet assessment, to discuss diet adherence/body weight maintenance and to supply bread products. A physician provided medical management. Parameters measured at 0, 12, 24 weeks were: blood glucose and insulin levels (via 8-hour day profiles) and fasting lipids. Analyses conducted were: plasma glucose by the glucose oxidase method (YSI model 27 glucose analyzer), HbA_{1C} (whole blood) via HPLC (BioRad Diamat), serum insulin by Pharmica Insulin RIA 100 radioimmunoassay kits, and plasma lipids via Sigma Diagnostic enzymatic kits. Except for plasma glucose analyses which were in triplicate, all other analyses were made in duplicate. Postprandial incremental area under the glucose and insulin curves (AUC) (Wolever and Jenkins, 1986) was divided into two 4-hour periods to evaluate separately the response to breakfast (peak #1) and lunch (peak #2). All dietary, clinical and biochemical data for the two study periods were subjected to analysis of variance (SAS GLM). Mean glucose and insulin response areas for 8-hour

profiles in both periods were compared (repeated measures analysis of variance). Data for study entry and both periods were compared using analysis of variance (SAS GLM). Correlations among dietary and biochemical data were computed.

During the study, body weight and triceps skinfold thickness of subjects did not change. Macronutrient composition of the diets remained constant. A mean dietary fat intake of 30% of kcals was maintained throughout the study. Amount of oat bran concentrate consumed was 71% of target (5.7 vs 8 bread exchanges per day). Oat bran concentrate bread/bread product intake increased mean total dietary fibre intake by 181%. Mean total dietary fibre intake increased from 18.5 g per day in the WB period to 34 g per day (9 g soluble fibre from oat bran concentrate) in the OBC period.

In the present study, the outstanding finding was the improvement in OBC period glycemic response after meals. OBC period total glucose incremental area under the curve was 41% ($p<0.05$) lower than in the WB period. After breakfast and lunch in the OBC period, glucose area under the curve was 35% ($p\leq0.01$) and 50% (NS) lower, respectively, than in the WB period. OBC period glucose excursions were reduced after meals; 14% after breakfast and 25% after lunch compared to the WB period. OBC period glucose peak values were lower than in the WB period; 13% for peak #1 and 15% for peak #2. The improvement in glucose values was associated with an amelioration in insulin values.

OBC period postprandial insulin response was reduced; total insulin area under the curve was 17% (NS) lower than in the WB period. After breakfast and lunch in the OBC period, insulin area under the curve was 22% ($p \leq 0.05$) and 13% (NS) lower than in the WB period. OBC period peak insulin values were lower than in the WB period; 15% for peak #1 and 11% for peak #2. OBC maximum insulin values were reached earlier than for the WB period; 26 min ($p \leq 0.05$) for peak #1 and 30 min for peak #2. In the OBC period, mean serum insulin values for peaks 1 and 2 were 15.3% and 11.0%, respectively, lower ($p \leq 0.05$ and NS) than in the WB period. For insulin, total area under the curve was negatively correlated ($p < 0.05$) with fibre intake, both total dietary fibre and soluble fibre. Earlier insulin peak values during the OBC period than the WB period suggest possible improvement in ß-cell function. Insulin response was not proportional to glucose response. A greater reduction in glucose response than insulin may indicate enhanced ß-cell sensitivity to insulinogenic stimuli. Possible mechanisms of action of oat bran concentrate (ß-glucan) are that the high viscosity of oat gum may slow glucose absorption in the small intestine and/or enhance insulin action at the cellular level. In addition, ingredients and preparation of the oat bran concentrate bread may have produced physical barriers and decreased exposure of the starch to digestive enzymes. No changes were found in fasting glucose or glycosylated hemoglobin levels.

In the OBC period, blood lipid values were altered favorably. OBC period mean plasma total-C and LDL-C were 14% and 23%, respectively, lower ($p<0.01$) than for the WB period. Mean HDL-C of subjects was similar in both periods. Blood lipid ratios improved; total-C/HDL-C and LDL-C/HDL-C ratios were lower by 14% (NS) and 25% ($p<0.05$), respectively, during the OBC than in the WB period. The decreased LDL-C/HDL-C ratio in the OBC period is of significance for the NIDDM individual, who is at high risk of cardiovascular disease. OBC period mean triglyceride was 5% lower than in the WB period and 10% lower than at study entry. Proposed mechanisms for the beneficial effect of oat bran concentrate on lipid lowering include the unique character of the oat bran concentrate, a slowed rate of lipid absorption, enhanced bile acid excretion, and/or effects of short chain fatty acids produced by soluble fibre fermentation in the small bowel.

Oat bran concentrate bread/bread products were well accepted long term; 75% were willing to buy commercially available products. Subjects reported satiety after oat bran concentrate bread product consumption and side effects were minimal. Use of oat bran concentrate bread and bread products as the dietary staple to increase fibre intake in the diet long term is feasible. Development of an acceptable oat bran concentrate bread containing 9 g soluble fibre made it possible to achieve an intake of 34 g total dietary fibre;

this level is in the range recommended for good metabolic control of diabetes.

In summary, results of the present study indicate that oat bran concentrate bread product consumption (9 g soluble fibre per day) significantly improved NIDDM subject's glycemic and insulinemic responses and reduced serum total cholesterol and LDL-C levels. Results showed a negative correlation between total and soluble dietary fibre intake and postprandial insulin response ($p \leq 0.05$). Further research is required to clarify the role of dietary fibres and other components in oat bran concentrate on glucose and insulin interrelationships and lipid lowering in diabetic individuals. It may be possible to improve metabolic control of non-insulin-dependent diabetes by dietary means. Oat bran concentrate bread products have practical potential to protect ß-cell function and to minimize long term complications of diabetes.

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APPENDIX

Appendix 1. Ethics Approval

University of Alberta
Edmonton

Faculty of Home Economics

APPENDIX 1

Canada T6G 2N8

115 Home Economics Building, Telephone (403) 492-3824
FAX (403) 492-7219

APPROVAL

FOR

PROPOSAL ON HUMAN RESEARCH

This is to certify that Dr. Zenia Hawrysh submitted a proposal for a research project entitled:

Effects of high fiber bread products on the metabolic control of Type II diabetes

to the Faculty of Home Economics Ethical Review Committee. The ethical criteria for human research have been met.

Date: June 25, 1991

Dr. T. Clandinin, Chair

Clandinin\Ethics mst

Appendix 2. Ethics Approval

University of Alberta
Edmonton
Canada T6G 2R7

Office of the Dean
Faculty of Medicine
2J2 00 WC Mackenzie Health Sciences Centre,
Telephone (403) 492-6621
FAX: (403) 492-7303

RESEARCH ETHICS BOARD

ETHICS APPROVAL FORM

Date: February 1992

Name(s) of Principal Investigator(s): Dr. E. Toth

Department: Medicine

Project Title: Effects Of High Fibre Oat Bread Products On Metabolic Control Of Non-Insulin-Dependent Diabetes.

The Research Ethics Board has reviewed the protocols involved in this project and has found them to be acceptable within the limitations of human experimentation.

Specific Comments:

Signed - Chairman of Research Ethics Board

for the Faculty of Medicine
University of Alberta

This approval is valid for one year.

\ethics\approve

Appendix 3. Letter to Potential Subjects

University of Alberta
Edmonton
Canada T6G 2M8

Department of Foods and Nutrition
Faculty of Home Economics
308 Home Economics Building, Telephone (403) 492-3831
Fax (403) 492-4821

June, 1992

Potential Subject,
#101 - 110 Street
Edmonton, AB
T0A 1T2

Dear Mr. Subject:

The Department of Foods and Nutrition at the University of Alberta is beginning a study which will look at the effects of oat fibre baked products on the metabolic control of men with Type II diabetes. The Metabolic Day Care Centre at the University of Alberta Hospitals is assisting us in recruiting participants for the study. We understand that you have been through the Metabolic Day Care Centre and fit the characteristics of patients that we are looking for for our study.

Please read the attached information sheet which describes the study in more detail. Would you think seriously about joining the study? Please note that your decision will not affect the quality of your continuing medical care.

We will contact you in the next week to discuss the study and answer any questions you may have. If you wish to reach us in the meantime, feel free to call Mary at 492-7674 (Evening: 425-3531).

Sincerely,

Mary Pick
Researcher

Appendix 4. Consent Form

INFORMATION SHEET

TITLE OF RESEARCH PROJECT: Effects of high fibre oat bread products on metabolic control of non-insulin-dependent diabetes.

INVESTIGATORS:

Dr. E. Toth	(492-6223)
Prof. M. Gee	(492-5031)
Dr. Z. Hawrysh	(492-3830)
Dr. M. Garg	(492-6991)
M. Pick	(492-7674)

PROJECT INFORMATION/PARTICIPANT INVOLVEMENT:

The purpose of this study is to evaluate the effectiveness of high fibre yeast bread and bread products on the long term blood glucose control and blood lipid levels of diabetics with NIDDM (Type II diabetes).

Participants in the study will be asked to:

1. participate in a long term study (a total of six months) involving regular daily intake of the test bread (standard white bread for three months; high fibre oat bread/bread products for three months) as part of their usual diet. A dietary program will be tailored to each participant's regular dietary pattern. Commercially baked bread/bread products will be supplied to the participants throughout the study.

2. participate in three evaluations of day blood glucose/insulin profiles in the Clinical Investigations Unit. This will require an overnight fast and consumption of meals typical of the period being tested (standard white bread or oat fibre bread). An intravenous catheter (IV) will be inserted, and twenty-one blood samples will be taken, at 30 minute intervals, for blood glucose and insulin profiles; an additional blood sample will be taken for other biochemical tests.

3. provide information about all the food, beverages, and supplements consumed to the graduate student. The participant will be asked to provide this information on eight occasions. Height, weight, and arm skinfold thickness measurements will be taken.

While no serious side effects are expected from consumption of the high fibre bread, some minor gastrointestinal discomfort, such as gas or flatulence, may be experienced on a temporary basis. During each of the three day blood/glucose profiles, some bruising or pain may be experienced around the IV site, and there is a slight risk of phlebitis (inflammation of the membrane of the vein). Less than 1/2 cup (100 mL) of blood will be drawn during each of the day profiles. This totals 1-1/3 cups (300 mL) of blood over six months, which is not harmful.

All records and documents relating to each participant are confidential, and no information that would expose his personal identity will be released or printed. A small honorarium ($100) will be paid to each participant upon completion of the study.

Appendix 4. Consent Form: continued

TITLE OF RESEARCH PROJECT: Effects of high fibre oat bread products on metabolic control of non-insulin-dependent diabetes.

CONSENT

I acknowledge that the research procedures described on the Information Sheet (above), and of which I have a copy, have been explained to me, and that any questions I have asked have been answered to my satisfaction. In addition, I know that I may contact the person named below, if I have further questions either now or in the future. I have been informed of the alternatives to participation in this study. I understand the possible benefits of joining the research study, as well as the possible risks and discomforts. I have been assured that personal records relating to this study will be kept confidential. I understand that I am free to withdraw from the study at any time and that this will not affect my continuing medical care. I further understand that if the study is not undertaken, or is discontinued at any time, the quality of my medical care will not be affected. I understand that if any knowledge gained from the study becomes available that could influence my decision to continue in the study, I will be promptly informed.

_____
Name

The person who may be contacted about the research is:

Dr. E. Toth Telephone: 492-6223

_____
Signature of Subject

_____
Name

_____
Signature of Witness

_____
Date

_____
Signature of Investigator or Designee

Appendix 5. Subject Profile

Clinical Study
Subject Information

Name _____ Date of Birth _____

Social Insurance No. _____ Duration of diabetes (known)_____

Medications _____

_____

Vitamin/Mineral Supplements? _____

Allergies (inc. foods) _____

Food Intolerance/ Dislikes _____

Do you follow your diet plan? Always _____ Usually _____

Sometimes _____ Never _____

Former Diets/Dietary Counselling _____

Do you exercise regularly? _____ How often? _____

Weight changes in the past year? _____

Do you have any gastrointestinal complaints? _____

Do you smoke? _____ If so, packs/day _____

Lifestyle Information

Occupation/Hrs. of work _____

Leisure/Physical Activities _____

_____

Religious/Ethnic Considerations _____

Family _____

Food Shopping: Who _____

What store _____

Food Preparation: Self _____ Other _____

Appendix 6. Dietary Intake Form

Clinical Study
48 Hour Dietary Recall

Subject:
Date:

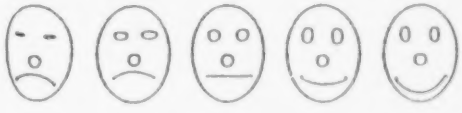
TIME hr. min.	PLACE #	SEQ ##	FOOD CODE	FOOD/DRINK	WORK AREA - Description, Amount	AMOUNT CONSUMED	FOOD UNIT

Appendix 7. Bread Acceptability Questionnaire

EFFECT OF OAT FIBRE BREADS ON DIABETES

Now that you have completed the oat fibre phase of the study, we would appreciate some feedback from you regarding the products: oat fibre bread, buns and muffins. Your response will help provide insight into the practical aspects of incorporating oat fibre concentrate into bread products.

1. Overall the oat fibre products were:



2. I liked ___ / disliked ___ the oat fibre bread because:

3. I liked ___ / disliked ___ the oat fibre buns because:

4. I liked ___ / disliked ___ the oat fibre muffins because:

5. If these products became available in the grocery store would you purchase them?

   Yes / No

6. List other products that you would like to see oat fibre incorporated in.

Appendix 7. Bread Acceptability Questionnaire: continued

7. Did you have any gastrointestinal discomfort (such as gas or flatulence, diarrhea) that you feel was related to the oat fibre products?

Yes / No

If yes, please elaborate:

8. Were you able to reduce your medication for your diabetes while on the oat fibre products?

Yes / No

If so, how much?

9. Did you experience a feeling of fullness while eating or after eating the oat fibre products?

If so, please elaborate:

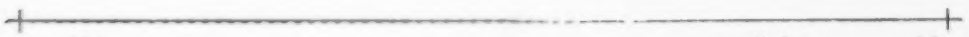
10. What other suggestions and comments do you have?

Appendix 8. Subject Questionnaire

OAT FIBRE - DIABETES STUDY

We would like to assess your overall feeling of wellness during the study. Would you please mark a spot on the following lines to represent how you felt before the study began and how you felt during each period of the study.

BEFORE THE STUDY:



felt terrible — felt very well

DURING THE WHITE BREAD PERIOD:

felt terrible — felt very well

DURING THE OAT FIBRE PERIOD:

felt terrible — felt very well

Appendix 8. Subject Questionnaire: continued

OAT FIBRE - DIABETES STUDY

Would you please check any of the following which apply to you during each phase of the study:

BEFORE THE STUDY	WHITE BREAD PERIOD	OAT FIBRE PERIOD
__tested blood sugars regularly	__tested blood sugars regularly	__tested blood sugars regularly
__yes	__yes	__yes
__no	__no	__no
if yes: __1X/day	if yes: __1X/day	if yes: __1X/day
__2X/day	__2X/day	__2X/day
__3X/day	__3X/day	__3X/day
__4X/day	__4X/day	__4X/day
__blood sugars well controlled	__blood sugars well controlled	__blood sugars well controlled
__yes	__yes	__yes
__no	__no	__no
__weight gain	__weight gain	__weight gain
__weight loss	__weight loss	__weight loss
__stable body weight	__stable body weight	__stable body weight
__felt well	__felt well	__felt well
__felt slightly under par	__felt slightly under par	__felt slightly under par
__felt poorly	__felt poorly	__felt poorly
__felt very poorly	__felt very poorly	__felt very poorly
__felt terrible	__felt terrible	__felt terrible
__energetic	__energetic	__energetic
__no energy	__no energy	__no energy
__tired	__tired	__tired
__GI discomfort	__GI discomfort	__GI discomfort
__GI gas	__GI gas	__GI gas
__felt bloated	__felt bloated	__felt bloated
__heartburn	__heartburn	__heartburn
__stomach cramps	__stomach cramps	__stomach cramps
__followed diet plan	__followed diet plan	__followed diet plan
__always	__always	__always
__usually	__usually	__usually
__sometimes	__sometimes	__sometimes
__never	__never	__never

COMMENTS:

Appendix 9. Subject Data

Study Entry Characteristics

Characteristic	Subject 009	Subject 011
Age (yrs)	54	49
Height (cm)	181	192
Weight (kg)	111.4	120.0
BMI[1] (kg/m^2)	34.0	32.6
HbA_{1C}[2] (%)	12.2	12.4
Plasma Glucose[3] (mmol/L)	21.26	18.21
Plasma Cholesterol[3] (mmol/L)	4.40	4.70
LDL Cholesterol[3] (mmol/L)	2.32	2.52
HDL Cholesterol[3] (mmol/L)	0.84	0.94
Plasma TG[3,4] (mmol/L)	2.70	2.70

[1] Body mass index
[2] Glycosylated hemoglobin
[3] Fasting values
[4] Triglyceride

Appendix 9. Subject Data: continued

Characteristics of Subject 009

Characteristic	WB Period	OBC Period
Age (yrs)	54	54
Height (cm)	181	181
Weight (kg)	110.4	110.0
BMI[1] (kg/m^2)	33.7	33.6
HbA_{1c}[2] (%)	13.0	13.5
Plasma Glucose[3] (mmol/L)	20.02	20.56
Plasma Cholesterol[3] (mmol/L)	5.70	5.00
LDL Cholesterol[3] (mmol/L)	1.51	2.52
HDL Cholesterol[3] (mmol/L)	0.84	1.10
Plasma TG[3,4] (mmol/L)	2.70	5.20

[1] Body mass index
[2] Glycosylated hemoglobin
[3] Fasting values
[4] Triglyceride

Appendix 9. Subject Data: continued

Characteristics of Subject 011

Characteristic	WB Period	OBC Period
Age (yrs)	49	49
Height (cm)	192	192
Weight (kg)	119.2	117.5
BMI[1] (kg/m^2)	32.3	31.9
HbA_{1c}[2] (%)	12.8	11.9
Plasma Glucose[3] (mmol/L)	16.71	9.42
Plasma Cholesterol[3] (mmol/L)	5.90	4.10
LDL Cholesterol[3] (mmol/L)	2.85	2.68
HDL Cholesterol[3] (mmol/L)	1.21	0.92
Plasma TG[3,4] (mmol/L)	4.00	1.10

[1] Body mass index
[2] Glycosylated hemoglobin
[3] Fasting values
[4] Triglyceride

Appendix 9. Subject Data: continued

OAT FIBRE - DIABETES STUDY

Summary of Diet Recalls: Average intake of nutrients per day calculated using four 48-hour dietary recalls in each dietary period

SUBJECT: 009 - JC

NUTRIENT	WHITE BREAD PERIOD	OAT FIBRE PERIOD
Total energy (Calories/day)	2250	3019
Carbohydrate		
grams/day	264	377
% of total Calories	47	49
Protein		
grams/day	105	135
% of total Calories	18	18
Total fat		
grams/day	89	111
% of total Calories	35	33
Cholesterol (mg/day)	482	470
Saturated fat		
grams/day	32	42
% of total Calories	13	13
Monosaturated fat		
grams/day	32	38
% of total Calories	13	12
Polyunsaturated fat		
grams/day	17	21
% of total Calories	7	7
Total Dietary Fibre		
grams/day	29	48

Appendix 9. Subject Data: continued

OAT FIBRE - DIABETES STUDY

Summary of Diet Recalls: Average intake of nutrients per day calculated using four 48-hour dietary recalls in each dietary period

SUBJECT: 011 - IB

NUTRIENT	WHITE BREAD PERIOD	OAT FIBRE PERIOD
Total energy (Calories/day)	2665	2551
Carbohydrate		
grams/day	355	354
% of total Calories	53	55
Protein		
grams/day	96	88
% of total Calories	15	14
Total fat		
grams/day	97	90
% of total Calories	32	31
Cholesterol (mg/day)	345	292
Saturated fat		
grams/day	41	29
% of total Calories	14	11
Monosaturated fat		
grams/day	37	34
% of total Calories	13	12
Polyunsaturated fat		
grams/day	15	20
% of total Calories	5	7
Total Dietary Fibre		
grams/day	18	42

Appendix 9. Subject Data: continued

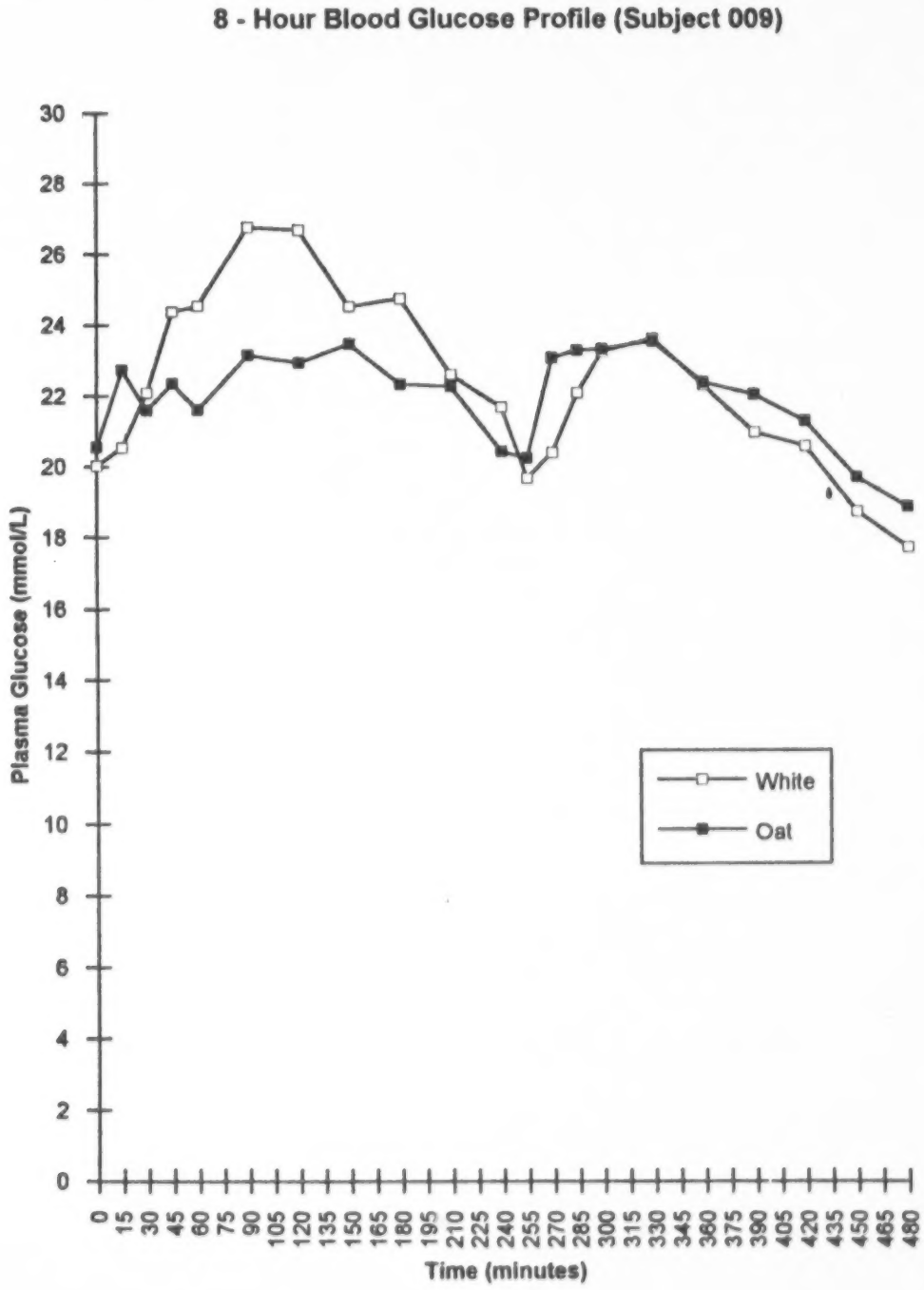
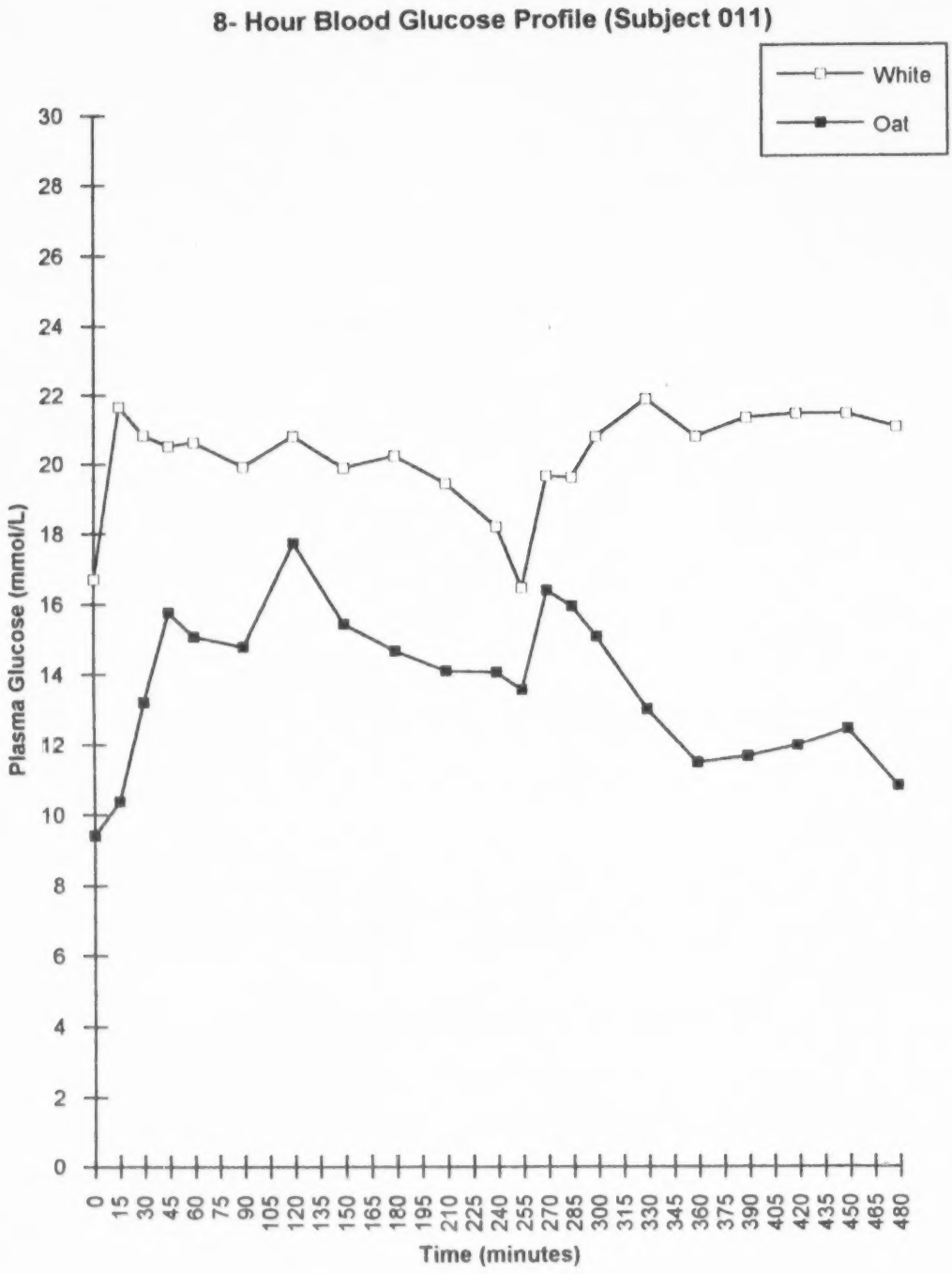
Subject 009 Glycemic parameters

Variable	WB Period	OBC Period
HbA_{1C} (%)	13.0	13.5
Fasting levels		
Plasma Glucose (mmol/L)	20.02	20.56
Glycemic response area		
• Glucose (mmol·min/L)		
total area (8hr)	727.4	485.3
part 1 area (4hr)	552.5	276.8
part 2 area (4hr)	174.9	209.9
Peak #1 response		
• Glucose (mmol/L)		
maximum	26.76	23.46
excursion	7.09	3.22
Peak #2 response		
• Glucose (mmol/L)		
maximum	23.61	23.54
excursion	6.74	2.90

Subject 011 Glycemic parameters

Variable	WB Period	OBC Period
HbA_{1C} (%)	12.8	11.9
Fasting levels		
Plasma Glucose (mmol/L)	16.71	9.42
Glycemic response area		
• Glucose (mmol·min/L)		
total area (8hr)	1159.24	1353.9
part 1 area (4hr)	500.1	556.5
part 2 area (4hr)	659.2	797.4
Peak #1 response		
• Glucose (mmol/L)		
maximum	21.65	15.76
excursion	2.23	5.22
Peak #2 response		
• Glucose (mmol/L)		
maximum	21.82	16.36
excursion	4.94	6.34

Appendix 9. Subject Data: continued



Appendix 9. Subject Data: continued